MAR 17 1925

Medical Lib.

Vol. LIV. MARCH, 1925 No. 2.

ARCHIVES OF OPHTHALMOLOGY

FOUNDED IN 1869 BY HERMAN KNAPP

EDITED BY

ARNOLD KNAPP AND WARD A. HOLDEN

NEW YORK

VASCULAR LESIONS OF THE RETINA IN YOUNG PERSONS HARRY FRIEDENWALD

THE SIGNIFICANCE OF PAPILLEDEMA TO THE NEUROLOGICAL SURGEON GILBERT HORRAX

PAPILLEDEMA IN OTO-LARYNGOLOGY LEON E. WHITE

THE SIGNIFICANCE OF PAPILLEDEMA TO THE OPHTHALMOLOGIST JAMES BORDLEY, JR.

ABRUPT MONOCULAR BLINDNESS, RESULTING IN COMPLETE OPTIC ATROPHY, DURING ENCEPHALITIS WITH DOUBLE PAPILLITIS. X-RAY OF OPTIC CANALS CLIFFORD B. WALKER

SERUM AND LENS-ANTIGEN EXTRACT TREATMENT FOR THE PREVENTION AND CURE OF CATARACT A. EDWARD DAVIS

ANGIOMATOSIS OF THE RETINA HUNTER W. SCARLETT

PRONOUNCED TRAUMATIC DISLOCATION OF EYEBALL WITHOUT DEMONSTRABLE FRACTURE OF THE ORBIT OR RUPTURE OF THE SCLERA—REPORT OF A CASE S. P. OAST

(Complete contents inside)

G. P. PUTNAM'S SONS

2 WEST 45TH ST., NEW YORK

PUBLISHED BIMONTHLY SUBSCRIPTION, SIX DOLLARS A YEAR

Entered at the Post-Office, New Rochelle, N. Y., as Second-Class Mail Matter

NOTICE TO CONTRIBUTORS

The editors and publishers of the ARCHIVES beg to offer some suggestions to authors who propose to favor them with the publication of their contributions.

1. Authors will receive gratuitously twenty-five reprints of their articles. If a greater number is desired,—**notice of which should be given at the head of the manuscript, or at the latest on the first proof,**—only the additional cost of press-work and paper will be charged to the author. The **number so ordered should be inclusive of the gratuitous copies.**

2. In preparing manuscript for the compositor it is requested that the following rules be adhered to:

a. Write on one side of the paper. Type-written MS. is preferred.

b. Write without breaks, *i. e.*, do not begin each sentence on a new line. When you want to begin a new line or paragraph at a given word, place before it in your MS. the sign ¶.

c. Draw a line along the margin of such paragraphs as should be printed in smaller type—for instance, all that is clinical history in reports of cases, etc.

d. Words to be printed in *italics* should be underscored once, in SMALL CAPITALS twice, in LARGE CAPITALS three times; **antique** type should be so marked.

e. Let the title of your paper indicate its contents. If it is a general title, for instance, Clinical Contributions, mention the subject of each special communication,—for instance: CASE I. Sarcoma of Iris; CASE II. Exostosis of Frontal Sinus, etc. These special titles will appear in the table of contents of each number and each volume, under the heading of the general title, so that they will not be overlooked.

f. Illustrations should be carefully drawn on separate sheets.

3. Authors receive proofs for revision, which they should correct and return without delay. We beg, however, to remind our contributors that changes in the copy are equivalent to resetting, causing so much additional expense. We therefore request them to make, if possible, no alterations at all in their MSS., or, at least, to limit them to what is of essential importance.

When sending manuscripts for the ARCHIVES OF OPHTHALMOLOGY, please address the editor, Dr. ARNOLD KNAPP, 10 East 54th St., New York City.

ARCHIVES OF OPHTHALMOLOGY.

VASCULAR LESIONS OF THE RETINA IN YOUNG PERSONS.[1]

A. WITH RECURRENT RETINAL AND VITREOUS HEMORRHAGES.

B. IN RETINAL DISEASE WITH MASSIVE EXUDATION (COATS'S DISEASE) INCLUDING VON HIPPEL'S DISEASE.

C. WITH CIRCUMSCRIBED EXUDATIVE CHORIO-RETINITIS.

BY DR. HARRY FRIEDENWALD, BALTIMORE.

(With three illustrations in the text.)

THE importance of retinal vascular changes in later life is now generally recognized. They are the objects of search and study, not of ophthalmologists alone but of internists as well. I am of the opinion that abnormal conditions of the retinal arteries and veins observed in the young have not received the same general attention. They are found in association with a number of well-known fundal diseases. My own interest in these conditions dates back a great many years. It is for this reason that I presume to lay before you a subject with which many of you may be quite as familiar as I. We shall consider the subject under the following heads.

1. Recurrent Retinal and Vitreous Hemorrhages.
2. Retinal Disease with Massive Exudation (Coats's Disease) including von Hippel's Disease.
3. Acute Circumscribed Exudative Chorio-retinitis.

[1] Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, October 16, 1924.

I. RECURRENT RETINAL AND VITREOUS HEMORRHAGES.

It was in cases of this condition that my interest in retinal vascular changes in the young was first aroused. Two such cases were reported in 1895[1]; at that time I did not know that similar observations had previously been made in 1881 by Eales,[2] suggesting disturbances in the retinal veins in these cases.[3] In my paper I described marked changes in the caliber of the retinal vessels and especially of the veins, constrictions, dilatations, engorgement, the formation of varices, and of newly formed vessels projecting into the vitreous. Subsequently many others reported similar findings and confirmed them by pathological examination.[4] I desire to present the subject to you by lantern slides of typical pictures found in literature and by the report of several cases.

The first slide is taken from Haab's *Ophthalmoscopy*[5] and shows retinal and subhyaloid hemorrhages and marked perivasculitis in one branch of the central retinal vein.

The second slide is copied from my article above referred to. In this case we have the development of definite varices and of vascular loops.

The third slide is taken from a paper by Arnold Knapp.[6]

The patient was a female, aged 25. Trouble with right eye for five years. Diagnosis of vitreous hemorrhages had

---

[1] *Journal Am. Med. Assoc.*, 1895, Vol. 25, p. 711. *Centralbl. f. prakt. Augenh.*, 1896.

[2] Trans. Seventh Internat. Med. Congr., Vol. 3, p. 51 (abstr.), published in full *Ophth. Review*, Vol. 1, p. 41.

[3] Eales had found the veins "large and tortuous," "the terminal branches of the retinal veins sometimes lost," "occasionally becoming suddenly tortuous like corkscrews" and he inferred that the hemorrhages occur from the venous radicles and from the capillaries.

[4] First by Fehr, *Centralb. f. Augenh.*, Vol. 25, p. 2. Leber has made a comprehensive survey of this subject in *Graefe-Saemisch Handbuch*, 2nd Ed. "Diseases of the Retina," Vol. 1, pp. 119–542.

[5] This illustration is found in the earliest edition of Haab's work published in 1895, in which it is numbered 27. It is described as Recurring Hemorrhage in Retina and Vitreous in a Young Individual. In later editions it is No. 35. These slides will not be reproduced in this paper since they are all easily accessible.

[6] *Arch. of Ophth.*, 1913, Vol. 42, p. 2.

been made. Patient in ill health. Brother tuberculous and a second brother died of tuberculosis. Right eye blind from total detachment. Left eye, no complaint but nerve congested. Two punctate hemorrhages at margin of disk and in periphery retinal veins show round areas about one third P. D. in diameter, evidently varices.[1]

---

[1] Since this paper was read, I have had the opportunity to examine a case analogous to that of Dr. Knapp is so far as a high degree of vascular disease was observed, with only small hemorrhages and with scarcely any disturbance of vision.

C. J. Z., male, age 13, was first seen in May, 1909, because of a blur of uncertain duration and some discomfort over his R. E. He had always enjoyed good health and had no history of serious illness. Vision R. E. was $\frac{6}{12}$ (?) and with $-.50$ sph, $\frac{6}{6}$ almost. V. L. E. $\frac{6}{6}$. The eye ground of the L. E. was normal and there was a prominent arterial loop over the disk; that of the R. E. showed disk of fairly normal appearance, somewhat hyperæmic, with fine connective tissue lying upon it and running out into the retina on the temporal side in fine lines to a denser mass of connective tissue lying about 1 P. D. away in an oblique direction. Beyond this the macular region showed a fine white infiltration with one darker spot, probably a linear hemorrhage, just above the fovea, and fine radial lines. Along the superior temporal artery there were clusters of intensely white spots, and in the far upper and temporal periphery a number of white spots and round hemorrhagic spots. No distinct vascular changes could be found. Other portions of the periphery showed no abnormalities.

The fields of vision, taken on several occasions, showed no abnormalities, and in particular, no central scotoma.

The boy was given a careful physical examination; his blood pressure was 110. The examination of the blood showed nothing abnormal excepting slight anæmia; urine normal. The patient was given iodide of strontium in five grain doses, t. i. d. He was kept under observation for a few months; after this he was not examined again until May, 1912, when the ophthalmoscopic conditions were very much the same.

Interested in learning what the further course of this case was, I sent for the patient in October, 1924 (after an interval of twelve and a half years), and he informed me that he had had no discomfort from the eye during these years. He had entered the army, had served throughout the war with the 113th Infantry, and since then had been very active in his business.

I was not a little surprised, on examining the eye grounds, to find the following:

V. R. E. $\frac{12}{18}$ poorly; with $-1.00 = .75$ at 135 $\frac{12}{18}$. His field of vision is quite normal, both for white and red; the blind spot is of normal size. V. L. E. $\frac{12}{18}$; the left fundus is normal. R. E. There is a faint veil of connective tissue which extends 2 diopters out in the vitreous; this veil extends over towards the macula about 1 P.D. in the form of fine horizontal

Another type of vascular disease is seen in the slides taken from a publication of Finnoff.[1] Fleischer[2] in 1914, published a very interesting case, shown in the next slides.

Male, aged 36. Hemorrhages of both retinæ with distinct vascular disease. On pathological examination marked tuberculous changes were found in the anterior portion of the eyeball and extensive tuberculous changes in the veins.[3]

---

streaks; at this point it is crossed by an oblique gray line, which appears elevated. Just beyond this region, that is to say, about 3 P. D. from the disk, is a circular area 1 P. D. in diameter, made up of numerous dots, chalky white in appearance. In the center of this area is a lobular hemorrhage and many fine hemorrhages are scattered about. This area is crossed by a small branch of the inferior macular artery. Further templewards, there are several patches of capillary dilatation, and one can see quite distinctly the whole capillary bed. In places there are sausage-like dilatation of the capillaries. The superior temporal vessel as it comes from the disk, appears reasonably healthy, but at about a distance of 3 P. D. from the disk, a larger hemorrhage overlies it and at this point it shows some variations in caliber; the inferior temporal artery here consists of two branches, one of which runs almost horizontally below the macula; there is a branch of this which goes across the area of white dots above mentioned. This vessel is quite tortuous; its sheath is everywhere visible, especially in small branches and shows great variations in caliber. The other branch of the inferior temporal artery is much less markedly diseased, but some of the smaller branches show great tortuosity. Templeward from the lesions described above, there is a small vessel running vertically, which shows a very large pear-shaped aneurysmal dilatation; the maximum thickness is about the width of the central retinal vein. This vessel is connected with either the inferior temporal vein or artery, but it is not possible to trace its connections definitely with one or the other.

The case described is remarkable for the reason that there is no history of profuse intraocular hemorrhages, but only in small ones as in the case of Dr. Knapp. It is remarkable in another respect because the vascular changes could not be found in the early examinations and evidently developed in later years.

[1] Finnoff, *Trans. American Ophth. Society*, Vol. 19, 1921, p. 243. Hemorrhages into retina and vitreous; also white exudate and marked vascular disease. Retinitis proliferans.

[2] *Klin. Monatsbl.*, Vol. 52, p. 768.

[3] The etiological importance of tuberculosis in the cases with periphlebitis of the retina, was first stressed by Axenfeld and Stock (*Graefe's Arch. f. Ophth.*, Vol. 66, p. 1, 1907, *Klin. Monats.* 1911 Vol. 49, p. 28. . . . *Klin. Monatsbl.*, Vol. 51, p. 71, *Bericht Oph. Gesells.* Heidelberg, 1920, p. 298.)

In 1915 Otori claimed to have found tubercle bacilli in the retina (?)

Jackson and Finnoff and others have accepted completely the tuberculous nature of the vascular changes just described and have published a number of interesting cases[1] and illustrations, showing various forms. No other satisfactory explanation has been proposed.

To these cases I desire to add the following.

CASE 1.—Miss S., otherwise healthy, complained on April 28, 1911, that her left eye had been blurred for two weeks. The retinal veins especially the inferior branches were engorged and tortuous but also showed marked constrictions here and there. Many were covered by a white exudate, which accompanied the vessels over large stretches into the farthest periphery. In some regions the vessels were completely obscured and converted into white bands. There were small and large hemorrhages in different parts of the fundus, but most profuse in the inferior portion. The arteries nowhere showed evidence of disease. Right fundus quite normal. There was improvement in her condition. In January, 1912, she returned with fresh hemorrhages especially in the lower nasal periphery.

CASE 2.—Mr. McC., aged 27. This patient, a medical student, was first examined in February, 1921, when he complained of headaches from studying. There was some ocular congestion, both conjunctival and retinal. There was a moderate hyperopic astigmatism, after the correction of which, his vision was perfect in both eyes. Two months later he returned complaining of a blur in the lower part of the field of vision of his right eye, which had come on a week before. Ophthalmoscopic examination showed a healthy optic disk and normal conditions except in the upper quadrant. The superior vein appeared healthy on and near the disk, but at a point about 3 P. D. from the disk, it was flanked and partially covered by a cloud-like effusion. At this point the vessel appeared slightly constricted, beyond it was wid-

---

(*Arch. f. Augenh.*, Vol. 79, p. 44). See also Finnoff (*Trans. Am. Oph. Soc.*, 1923, p. 87.)

Leber (*loc. cit.*, p. 549) states in his comprehensive review of this subject that "there is unquestionable relation between these conditions and tuberculosis."

[1] Jackson, *Trans. Am. Oph. Society*, 1916, Vol. 14, p. 577.
Jackson and Finnoff, *Am. Journ. Ophth.*, 1920, Vol. 2, p. 715.
Finnoff, *Trans. Am. Oph. Society*, 1921, Vol. 19, p. 243.

ened. Scattered along various tributaries of this vein were small white patches covering the vessels with very small petechiæ in the neighborhood. The retinal artery and its branches even in the region described appeared healthy.

General medical examination of the patient revealed the following. History of typhoid, pneumonia, pleurisy, and acute rheumatic fever with pericarditis. The latter has since recurred. No evidence of tuberculosis. Wassermann reaction negative. Of the possible foci of infection only the tonsils were suspicious. They were, therefore, removed.

During the following months the white spots disappeared but the hemorrhages became more numerous and extensive, and finally there occurred a series of large hemorrhages into the vitreous during a period of almost three years. At intervals between these hemorrhages the vitreous would clear sufficiently to allow a view of the fundus. The picture of retinitis proliferans gradually developed, heavy strands of connective tissue extending throughout the upper half of the fundus. At the last examination, May 25, 1924, in addition to the proliferating retinitis a point of interest was the presence of loops of the retinal vessels rising from the level of the retina and extending into the vitreous, one as far as 4 D. (See Fig. 1.) The superior retinal vein could not be fol-

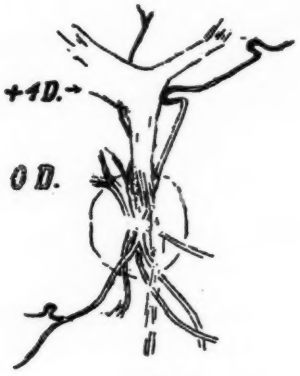


FIG. 1.

lowed more than 2 P. D. from the disk. From here on it was lost under an extensive branching mass of connective tissue. Vision which had at one time been reduced to bare light perception, was now $\frac{1}{5}$. The visual field showed an enlargement of the blind spot and a marked loss in the lower

and temporal regions. The defect extended almost but not quite to the blind spot.

CASE 3.—Thomas H., aged 10, was first seen October 9, 1909, for a slight asthenopia. October 21, 1917, V. R. E. $\frac{20}{15}$, V. L. E. $\frac{20}{15}$. Perfect vision, slightly better in the left eye. On October 21, 1918, he came complaining that a cloud had appeared before the left eye two days before. Two weeks before he had been ill with grippe.

He had had the usual diseases of childhood; his tonsils and adenoids had been removed. He had at one time had a slight endocarditis, which passed without leaving any noticeable impairment. He was examined with special reference to tuberculosis, but with negative results.

The left fundus was very hazy, there were profuse retinal and vitreous hemorrhages in the lower portion of the fundus. There was some history of a blow on the eye before the haze appeared.

As the blood was absorbed, whitish masses appeared in their place. His vision cleared up and remained perfect until he had another attack of grippe with a bad cough in the early part of February, 1921. He had been confined to bed for a week; when he was able to leave the house, he walked down town and noticed a blur before the left eye. I saw him about a week later and found a recurrence of the hemorrhage in the same eye, again in the lower periphery, the hemorrhage presenting a long, comet-like shape.

There were also the old opacities which resulted from the previous hemorrhage. There were no ophthalmoscopic indications of vascular disease, but in the nasal and inferior portion of the fundus, I found a branch of the inferior nasal

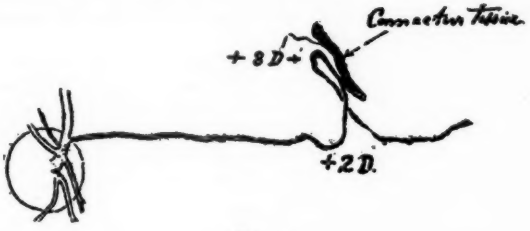


FIG. 2.

vein leaving the niveau of the retina and extending out into the vitreous to the connective tissue mass, where it formed a loop. (See Fig. 2.) The hemorrhage gradually disappeared.

I feared the possibility of detachment of the retina and have watched the case from time to time, but no trouble has developed. The central vision is quite normal, and the field of vision shows nothing wrong except an indefinite but small scotoma far out in the lower temporal quadrant.[1]

Another case presenting this curious vascular formation, is the following, which has many other features of interest. It was published in 1895 in my paper referred to above.[2] I had the opportunity of examining the patient again in 1923, after an interval of almost 29 years and wish to report on the final outcome.

CASE 4.—Mr. K. When 15 years old, the patient suffered from retinal and vitreous hemorrhages. Among the numerous vascular lesions described at that time the ones of most interest to us now, in view of subsequent developments are those in connection with the inferior nasal vein. In January, 1893, it was noted that this vessel at a point about 2 P. D. from the disk was covered by a small bluish cloud, while at a point 4 P. D. from the disk the vessel suffered a marked constriction. Two months later it was noted that at "about 2 P. D. from its origin" the vein "sent off a large number of fine capillary vessels which formed a meshwork in the vitreous. At this point there was the appearance of a bend or twist in the vessel. A short distance beyond these were two thickenings which upon close examination were seen to be twists in the vessel. Beyond these points the vessel became exceedingly attenuated for about 1 to 2 P. D., then widened out and divided into fine twigs. . . ." Similar changes were seen in connection with other retinal vessels.

Three weeks later the following note was made. "The vascular veil arising from the vena nasalis inferior had greatly increased in size and now reached the lower inner margin of the papilla." Vision at this time was $\frac{6}{9}$ almost.

During the next year the patient had several large vitreous hemorrhages. At the examination in April, 1895, the vitreous had cleared sufficiently to permit ophthalmoscopic examination it was noted. "There are large masses of vascularized connective tissue in the vitreous, much larger than

[1] The patient was last seen on February 13, 1925. The looped vessel is unchanged; there have been no further hemorrhages and central vision is perfect.

[2] *Journ. A. M. A.*, 1895. See Case 1.

when last seen, presenting the picture of retinitis proliferans."

The patient was not seen again until November, 1923, a lapse of almost 29 years. From the history it is clear that he has had no more large vitreous hemorrhages, but the sight of the R. E. has slowly grown worse.

V. R. E. = $\frac{6}{9^{+}5}$. With $-$ 11.50 = $-$ 1.0 ax 180 V. = $\frac{6}{15}$. The emmetropia of the L. E. has remained unchanged. V. L. E. = $\frac{6}{4}$ emmetropia, beginning presbyopia.

R. E. Ophthalmoscopic examination showed slight lenticular opacities, optic disk of good color, large conus. The macular region appears normal. *Nowhere any signs of retinitis proliferans.* The inferior nasal vein at a point about 2 P. D. from the disk abruptly turns off into the vitreous, reaches a small floating connective tissue mass at a point which can be seen with + 12D. Here it forms a loop returning back to the retina, one or two vessels breadth from the point of its leaving. The ascending and descending portions are in close contact often intertwining. There is no evidence of a fold in the retina at this point. Nearby vessels pursue their course undisturbed. Just beneath the pedicle is an area of choroidal atrophy. (See Fig. 3.)

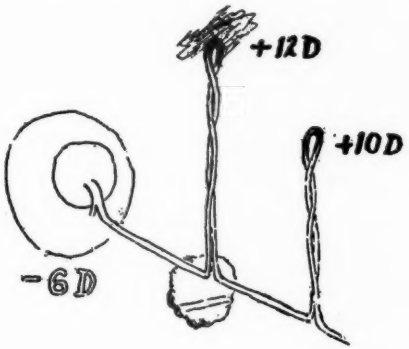


FIG. 3.

Following the vein about 2 P. D. farther peripheralward we come to another similar loop which extends into the vitreous to a point visible with + 10 D. This loop is not capped with a dense white mass but with a very filmy vitreous opacity. The arteries are all somewhat narrow. Otherwise the vessels show no abnormality. There are several

areas of circumscribed choroidal atrophy and several more diffuse areas of pigmentary disturbance.

In this case we have been able to observe the disappearance of the masses of fine new formed blood-vessels, the cessation of hemorrhages and the development of the large vascular loops, which have evidently not produced detachment of the retina. The absence of retinitis proliferans is unexpected. There is another change that has occurred—the development of a high degree of myopia in the affected eye—a change that it would be difficult to explain.

I desire to add another case in which I have had the rare opportunity of studying this disease during an unusually long period, a case that has likewise presented many points of special interest.

CASE 5.—Mrs. K. From the time of her marriage in 1885, when she was 18, she had recurrent retinal and vitreous hemorrhages. The intervals were of very variable lengths. My earliest notes are dated May, 1894, when she complained of spots before the eyes. There were numerous vitreous opacities, some large, in the right eye. In the upper temporal portion there was a choroidal scar to which large vitreous opacities seemed to be attached. In the left the vitreous was clear. From the superior temporal vessels there was a new formation of very fine blood vessels sprouting out into the vitreous in the form of minute loops. March 14, 1895—fine vessels over the left optic disk springing from the upper nasal vessels as they leave the disk. In the temporal periphery a large number of very tortuous tufts of fine blood vessels. On two of these, large rounded red bodies which appear to be varices. May 26, 1895—beginning of connective tissue formation in the temporal periphery of the left eye in the region of the new vessel formation, beginning of retinitis proliferans. The tufts of newly formed blood vessels grew larger and the retinitis proliferans gradually became more marked. Slight hemorrhages from time to time. On February 21, 1896, there was a profuse hemorrhage in the left vitreous. August 5, 1896, at 8.30 P.M. the patient came to me complaining that a hemorrhage had just occurred. Streaks of blood were to be seen and fine lines with a little drop at the end of each jutting out into the vitreous from the region of the papilla. These were about three or four in number and it was evident that they were supplying the fresh mass of blood some

distance below the papilla. One could distinctly see the gradual accumulation of blood in the form of the drops mentioned, which showed that the hemorrhage was still going on.[1] The last massive hemorrhage occurred in May, 1900.

May 31, 1923, the patient returned complaining of spots before her left eye. The optic disk was pale and the vessels showed some narrowing. In the temporal periphery there was a large mass, retinitis proliferans. In the lower periphery there were several hemorrhages and in the upper temporal periphery, a large one. There was one clump of very badly diseased blood vessels in the temporal region with miliary aneurisms. Vision was almost perfect in both eyes.

During the following summer, she developed diplopia and exophthalmus of the left eye without change of central vision. When I saw her in September, there was marked limitation of movement with an exophthalmus of about 4*mm*. Subsequently she developed involvement of the vertibræ probably due to a metastasis, but thus far the primary growth has not been discovered. It is needless to say that every form of examination had been applied. The patient was put under radium treatment. The degree of exophthalmus fluctuated and on the whole has not increased. The last fundal examination was made March 19, 1924, and there were no changes except that no hemorrhages could be found. At the present time nodular masses can be felt which cause protrusion of the left lower lid. The general condition indicates that the patient can live but a short time.

From the cases pictured and described it will be seen that the vascular disease is of three main types; first the perivasculitis which appears at an early stage and which disappears later; and second the changes in caliber and the development of varices, tufts of fine blood-vessels protruding into the vitreous, which likewise tend to disappear and third great loops of large vessels which jut out into the vitreous and seem to be late and permanent changes.

I have not been able to find any descriptions of such loop formation. At a meeting of our local Society in Baltimore, in 1920, I demonstrated the only other case which has come under

[1] I know of but one similar observation. Dufour and Gonin observed bleeding from a retinal vein. Cited by Leber, *loc. cit.*, p. 544.

my observation.[1] It was seen in a young negro suffering with profuse retinal hemorrhages. The only reference that may possibly have a bearing on this subject is found in Leber's work."[2] "In cases of formation of connective tissue in front of the retinal in the so-called retinitis proliferans, . . . partial detachment of the retina may occur, whereby the retinal vessels move forward, and thus it may become difficult to distinguish between these and the new-formed vessels, etc." In none of the cases that I have described has there been any evidence of retinal detachment and in all of them the vessels that formed the loops could be followed with the greatest ease, the media being quite clear. In appearance they were not unlike the congenital loops that spring from the disk,[3] except that generally the highest peak of the loop is close to a small or large mass of connective tissue in the vitreous.

The last two cases (Cases 4 and 5) prove that the final outcome of this type of disease is less unfavorable than generally assumed; that the hemorrhages tend to cease and that good vision may be regained and retained.[4] The long course during which these cases have been followed, has made it possible to determine, (a) that the vascular lesions were limited to the eye, for no evidence of other vascular disease was ever found; and (b) that the period during which the hemorrhages occurred, was limited, which justifies the assumption that the vascular lesion was not congenital, but was a temporary disorder due to unknown causes and only the consequences of these lesions are permanent.

2. RETINAL DISEASE WITH MASSIVE EXUDATION.

These cases of retinal disease first grouped under the above heading by Coats in 1908[5] present in most cases quite characteristic vascular changes in the retina. In 1914 I reported

---

[1] *Am. Journ. of Ophth.*, Vol. 3, p. 224.

[2] *Graefe-Saemisch Handbuch*, 2nd Ed., Vol. 7, page 130.

[3] Leber, *loc. cit.*, p. 78.

[4] Leber says: Finally even after a period of years, *tolerable* vision may be retained, *loc. cit.*, p. 544.

[5] Royal London Ophth. Hosp. Rep., Vol. 17, p. 440 and *Graefe's Arch. f. Ophth.*, 1912, Vol. 81, p. 275.

four cases of this disease and published two colored drawings which I here present in slides, together with the plate from *Oellers Atlas*, showing "miliary aneurisms."

Since that time I have had the opportunity of observing two additional cases, one of which has been published.[1]

The second case is the following:—

CASE 6.—Vincent D., aged 5, was seen through the courtesy of my colleague, Dr. Crouch, April 17, 1923, and again May 23, 1923. The child had been brought on account of convergent strabismus.

The left eye was normal and the right eye showed a large area embracing the macular portion and extending below it and to the temporal side and especially far up and over the disk studded with small white spots. The macular region itself contained a large gray mass in which there were fine brilliant crystals. The region therefore specially affected was that supplied by the superior temporal vessels, though it also extended to a slighter degree along the inferior temporal vessels. The nasal portion of the fundus appeared fairly healthy. Separated entirely from these lesions there was in the temporal periphery a large gray surface having the appearance of retinitis proliferans, and on it there were large numbers of very dilated vessels which showed distinct aneurismal dilatations, some quite large; this picture could be followed to the outermost periphery.

The child was last seen in January, 1924, and the picture had not changed.[2]

I may add that Dr. Crouch had the general condition of the child carefully investigated; Wassermann and Von Pirquet were negative.

This is the youngest case of retinitis with massive exudation which I have had the opportunity to examine.

The vascular anomalies which characterize these conditions are.

a. Tortuous and engorged veins,

---

[1] *Trans. Am. Oph. Society*, 1918, Vol. 16, p. 216.

[2] Since reading the paper the patient was examined again, November 26, 1924. The ophthalmoscopic picture was much changed. The white spots around the macular region had in great part disappeared and the evidences of vascular disease in the temporal region were likewise much less marked. In the inferior temporal region, there was an increase of the white exudates with an area of elevation, grayish in color.

B. Aneurismal dilatations,

C. Sclerosis of the vessels and perivasculitis.

Coats ascribes these conditions to vascular disease and consequent hemorrhages, often profuse, into the external layers of the retina and their subsequent organization, together with degenerative changes in the retina.

Rodos,[1] who examined a very early case pathologically found extensive vascular changes, sero-fibrinous infiltration of the retina and the formation of a layer, rich in cells and vessels, containing pigment epithelial cells, and firmly adherent to the retina, lying between the retina and the choroid.

Mention should be made, however, of the fact that Coats reported cases which showed no marked vascular changes, ophthalmoscopically.

Appearances very similar to these are found in other and very different conditions. Von Hippel[2] in 1904 described a curious vascular tumor formation, an *angiomatosis retinæ*, with disease of the retinal vessels, degenerative changes in the retinal nerve elements, hyperplasia of the glia and sub-

---

[1] *Graefe's Arch. f. Ophth.*, Vol. 105, p. 973.

[2] Von Hippel, *Graefe's Arch. f. Ophth*, 1904, Vol. 59, p. 83, *ditto* 1911. Vol. 79, p. 350, *ditto* Vol. 95, p. 173. The first one to describe this condition pathologically was Treacher Collins, *Trans. Ophth. Soc. United Kingd.*, Vol. 14, p. 141.

Other references are as follows:

Meller, *Graefe's Archiv*, Vol. 85, p. 255. He regards the Gliosis as the primary factor, the vascular formation as secondary.

Ginzburg and Spiro, *Graefe's Archiv.*, Vol. 88, p. 44.

Emanuel, *Graefe's Archiv*, Vol. 90, p. 344.

Bergmeister, *Graefe's Archiv*, Vol. 105, p. 1.

Case of Microphthalmus which showed changes analogous to Angiogliomatosis.

Brandt, *Graefe's Arch. f. Ophth.*, Vol. 107, p. 127. He tries to show that the growth is a hemangioendothelioma.

Berblinger, *Graefe's Archiv*, Vol. 110, p. 394. He regards the condition as a capillary hemangioma.

Knape, *Arch. f. Augenh.*, Vol. 60, p. 41.

Leidel, *Ophthal. Gesellsch*, 38 Meeting, p. 335.

Gamper, *Klin. Monatsbl.*, Vol. 61, p. 525. He considers the condition as due to a congenital teleangiectasis of the retina and finds the clinical and pathological changes identical with those of retinitis with massive exudation.

Ditroi, *Klin. Monatsbl.*, Vol. 59, p. 43.

retinal hemorrhages with subsequent organization. I have not met with any cases of this type but for completeness sake shall show one such drawing, that of Wood.[1] This case was a

> Female, aged 50. For a number of years had enlarged retinal veins. Complained for a few days of failure of sight in right eye due to hemorrhages in the vitreous. A large tumor was found in the upper part of the fundus, with enormously enlarged veins. Vision later returned to normal.

A similar picture was reported by Knape,[2] but was due to an entirely different cause.

> The patient was a boy, aged 10. Blind in left eye for four weeks. Described as "Von Hippel's Disease, Retinitis proliferans." On microscopic examination, glioma of the retina was found.

We are forced to the conclusion that these conditions, so similar in ophthalmoscopic picture, may result from very different underlying lesions.

3. ACUTE CIRCUMSCRIBED EXUDATIVE CHORIO-RETINITIS.

In this affection, retinal vascular disease is not infrequently observed. In the well-known illustration of Haab,[3] thrown on the screen, a case is pictured in which "an arterial branch running toward the macula reveals a whitish alteration of its walls."

In my paper[4] on this subject in 1902, I first emphasized this complication, calling attention to the frequently accompanying changes in the retinal blood-vessels. I described the vascular affection as a perivasculitis, showing itself in numerous white spots arranged along the sides or upon the surface of the vessels. An illustration which accompanied the paper is shown on the screen.

---

[1] H. Wood, *Trans. of Ophth. Society of United Kingdom*, Vol. 29, p. 115.

[2] F. Knape, *Arch. f. Augenheilkunde*, Vol. 60, p. 96.

[3] *Ophthalmoscopy*. First German edition published in 1895, fig. 55a. In 2nd Am. ed., fig 73a. In 3rd Germ. ed., fig. 67a.

[4] *Trans. Am. Ophth. Soc.*, 1902, Vol. 9, p. 577.

In a recent paper[1] I pointed out that in a later series of cases vascular diseases was also found in a number of cases. These were very varied. Perivascular lines, whitish effusion along the sides or on the surface of the vessels, marked localized constrictions, or dilatations of the veins, venous engorgement.

This subject has apparently not been given any special attention. Thus I have been able to find very few illustrations depicting the condition. I shall throw but one more slide upon the screen, one taken from Hepburn.[2] I desire to report a number of cases which will indicate its importance.

CASE 7.—Mr. G. J., aged 19. March 22, 1922. L. E., extensive irregular area of choroidal atrophy in the center with some pigment. Visual trouble since 1914. R. E. Acute chorio-retinitis on the lower nasal border of the disk. In the macular region there are a number of very small whitish spots along a macular vessel.

CASE 8.—Mr. H., November 7, 1908. Choroidal lesion along the course of the inferior macular artery. Paracentral scotoma. Numerous whitish effusions along course of the artery.

CASE 9.—Mr. H., March 5, 1918. Exudative chorio-retinitis in the inferior periphery of the fundus. The fundus shows certain rather anomalous and very small masses of exudate along the blood vessels. These are seen best in the nasal half of the field arranged like beads along the course of the vessels.

CASE 10.—Miss E., September 22, 1919. Fresh lesion ten days' duration. Dense effusion over the nasal part of disk with adjacent mass of pigment. The superior nasal vein is irregular in its caliber and the superior nasal artery somewhat covered with effusion. December 1, 1920, fifteen months later. Connective tissue mass over the center of

[1] Read before the *Oxford Congress of Ophth.*, July, 1924.

[2] Hepburn, Inflammatory and Vascular Diseases of the Choroid. *Trans. Ophth. Society of United Kingdom*, Vol. 32, 1912, p. 366.

Colored drawing showing deep localized choroiditis with evident disease of an artery passing over it. This case is also pictured by Parsons as under title of Metastatic Choroiditis. (Diseases of the Eye, 4th ed., 1923, p. 319.)

the disk toward the nasal side. Pigment disturbance along the entire nasal side and nasal lower side extending out into retina. The superior nasal artery is bordered by a marked whitish line on each side. The inflammation has entirely disappeared and the media are perfectly clear.

CASE 11.—Mr. V., aged 24. March 24, 1924. Spots before L. E. for a week which in the course of a few days developed into a veil. The lesion lies directly over the main branch of the central vein which runs down a little and then nasally almost horizontally just below the median line. About 3 P. D. from the disk the first large mass is seen; this is about six vessel thicknesses wide and three or four high, and is surrounded by an extensive haze. A little further nasally the second very small mass is seen. The vein itself changes caliber frequently, becomes at times almost threadlike and then wider again. Vessels in the neighborhood show similar changes; thus, some fine vessels which are seen in the fairly clear retina a little to disk side of the inflamed area, show decided peri-vascular lines.

April 9, 1924. Left Eye—Lesion has grown smaller; much less marked. There are a number of more intense white spots in the area affected; a nasal vein is partly covered by infiltrates at a number of points so that it appears as though there were slight contractions and breaks.

I desire to point out again that the vasculitis is of just the same appearance, ophthalmoscopically, as that described above in cases of retinal and vitreous hemorrhage in the young.

The close relationship between these conditions is also seen in the

ASSOCIATION OF ACUTE CIRCUMSCRIBED EXUDATIVE CHORIORETINITIS AND HEMORRHAGE INTO RETINA AND VITREOUS.

I have elsewhere referred to the association of these conditions. The following cases are examples of the mild and the severe forms:—

CASE 12.—Mr. W. S. aged 20. October 22, 1920. Suffers from attacks of ophthalmic migraine. On the day previous had such an attack which left a blur before the R. E. The retinal vessels were congested. Area of fine inflammation below the disk with haze and linear striation in the macular region. In the upper nasal part of field about 3

P. D. from disk large area of fresh exudation. In this region the blood vessels were exceedingly fine and thread-like. Irregularity ot pigment in the temporal periphery. The boy's father was suffering from advanced tuberculosis. Patient himself showed no active lesion. Tuberculin treatment was begun.

November 24, 1920. Small retinal hemorrhage. Veins enlarged to at least twice their normal caliber.

December 16, 1920. Two fresh retinal hemorrhages.

March 23, 1921. Inflammatory effusion has almost disappeared. Central vision normal.

September 10, 1923. After a long interval, careful ophthalmoscopic examination was made. Cornea and vitreous clear; optic disk normal in color and blood vessels healthy; but there is a faint connective tissue veil extending in the form of a narrow band of fine fibers upwards and inwards from the surface of the disk, gradually broadening out to a point about 3 P. D. up and in where there is an extensive area of choroidal atrophy and pigment accumulation; an irregular, zigzag vessel seems to spring from the very center of this area, unconnected with any nearby vessels, and goes out as the superior nasal vein. In the temporal periphery, there are some slight pale spots of old choroiditis. Vision is perfect.

CASE 13.—Miss H., aged 18. November 19, 1921. First seen on account of severe chorio-retinitis with slight iritis and extensive retinal hemorrhages in the left eye. She presented an old atrophic patch near the macula in the R. E. She had had her badly infected tonsils removed 3 years before. Wassermann negative. Evidence of an apical tuberculous focus. The patient was a sufferer from ophthalmic migraine. Milk injections and later tuberculin treatment was instituted. There were recurrence of hemorrhages so that in February, 1922, her vision fell to $\frac{8}{200}$.

In May, 1922, vision had returned to normal but the disk was markedly obscured especially on its nasal half by the development of connective tissue extending out into the vitreous. In the meantime the R. E. had not undergone any changes. There was pallor of the macular half of the disk and an area of old choroidal scarring between the disk and the macula. The central vision was perfect. On April 20, 1923, the vision of the R. E. became blurred and on April 26th, it was reduced to $\frac{20}{100}$. The central vision continued to grow worse, the vitreous became cloudy, the disk

[1] For reference to similar cases see Feingold, *Am. Journ. Ophth.*, 1920, Vol. 3, p. 499.

hazy and the macular vessel that passed into the region of the old scar, became occluded and converted into a white line. A fresh effusion developed and within a few days (May 10th) deposits were seen on the membrane of Descemet. There was now a complete loss of central vision and scotoma of about 20 degrees in each diameter. The effusion gradually cleared up but without improvement of vision and without alteration in the appearance of the occluded vessel.

The cases just reported suggest that the same cause may be productive of both conditions and that the presence of similar vascular disturbances especially periphlebitis may be the indication of this close relationship. Those who like Fleischer regard the vascular disease as tuberculous find in it a ready explanation. But more evidence is required before this assumption can be accepted as proved. For while it is alluring to attribute many of these conditions to tuberculosis, all will agree that a clear and indubitable diagnosis of the tuberculous character of these affections is most difficult.

CONCLUSION.

Disease of the retinal vessels is a frequent accompaniment of a number of pathological conditions of the fundus. We must note, however, that the vascular lesions that are seen with the ophthalmoscope are sometimes at a distance from the gross retinal lesion. In some conditions the vascular disease is the cause, as in the hemorrhages into the retina and vitreous and probably in the deep hemorrhages of retinitis with massive exudation. In other cases the vascular conditions may be the result of primary retinal disease producing inflammatory and obstructive affections of the vessels. We do not know whether the primary vascular disease is at times congenital or under what conditions it develops when acquired, but tuberculosis is believed by many to play an important part.

For those who are fond of finding names for such conditions as described, *Juvenile Retinal Angiopathy* as suggested by Cords[1] may be satisfying but it does not help us to a better understanding of the conditions.

[1] *Zeitschr. f. Augenh.*, 1911, Vol. 26, p. 441.

THE SIGNIFICANCE OF PAPILLEDEMA TO THE NEUROLOGICAL SURGEON.[1]

By Dr. GILBERT HORRAX, Boston, Mass. Associate in Neurological Surgery, Peter Bent Brigham Hospital.

IN attempting to arrive at proper conclusions concerning the significance of papilledema to the neurological surgeon, I have used the avenues of approach which were at my disposal, with the object in mind of answering the following questions. First. What conditions other than brain tumor or its surgical equivalent can cause choked disks? Second. What has been the previously assumed etiology of the known papilledema in a considerable series of verified brain tumors, together with the various forms of treatment employed for relief? Third. What harm, if any, to the patient has resulted from the delay incident to treatment directed upon an erroneous assumption?

To answer the first question a study was made of all cases which have been registered on the surgical service at the Peter Bent Brigham Hospital with the diagnosis of a condition which either actually did or conceivably might cause choking of the disks. For questions two and three a series consisting of the last one-hundred cases of verified brain tumors showing papilledema was investigated. These cases were in no way

[1] Read with the next succeeding papers by Drs. White and Bordley and a paper by Dr. W. R. Parker on "The Mechanism of Papilledema," published in the *Archives of Neurology*, at the Montreal Meeting of the American Academy of Ophth. and Oto-Lar., Dr. Walter B. Lancaster, President, constituting a "Symposium on Papilledema," Sept. 17, 1924.

selected, and merely represented the most recently admitted patients having the conditions indicated.

Concerning the first problem, it was found that during the past ten years there were 183 patients other than those with brain tumor (including abscess, gumma and tuberculoma) whose diagnosis upon discharge represented a condition which we know may cause papilledema. Of these patients, however, there were 44 who had conditions which we may term "tumor equivalents." By this is meant conditions simulating brain tumor, for which an intracranial operation is indicated just as positively as in tumor. The great majority of these, namely 33, were patients suffering from chronic or subacute cisternal arachnoiditis. This condition has been called by different names, such as serous or cystic meningitis. Its etiology is uncertain, but may possibly be a cerebral infection allied to poliomyelitis. At all events, it produces a thickening of the arachnoid membrane with a consequent backing up of cerebrospinal fluid in the large arachnoidal cisternæ, especially along the base and around the cerebellum. This fluid accumulation simulates tumor so exactly that it cannot be differentiated with certainty except by an exploratory operation. Such an operation, we believe, is always indicated because the condition is quite curable by this means, and although in some instances the choked disks might subside spontaneously, nevertheless they do not do so in all cases and the patient's vision should not be subjected to that hazard. The other two conditions of "tumor equivalents" were oxycephaly (8 cases) and pachymeningitis hemorrhagica interna (3 cases). The former all had a low grade of papilledema with slow, gradual formation of new tissue, and it is our belief that they should be decompressed in order to relieve the slight but constant intracranial tension and thus preserve the optic nerves from further damage. The three patients with pachymeningitis also had choked disks and were all cured by a complete mechanical removal of the organizing blood clot. These 44 therefore should be aligned with the tumor cases, leaving a balance of 139 patients in the group other than tumor or its equivalent. These 139 cases represented seven conditions divided into the numbers shown in Table I.

TABLE I.

Diagnoses	*Number of Cases*	*Number with Papilledema*
Sinusitis (all kinds)	52	1
Arteriosclerosis	36	4
Encephalitis	25	1
Tuberculous meningitis	9	3
Meningo-encephalitis	9	0
Intracranial aneurysm	7	3
Cerebral thrombosis	1	1
	139	13

Of the total number there were 13 which were said to have shown choking of the disks, this being 9.4 per cent. Therefore it will be seen at once that an extremely small percentage of the conditions other than tumor had actually caused papilledema in the series at the Peter Bent Brigham Hospital. When this figure is compared with the percentage of tumor cases causing choked disks, a percentage acknowledged to be large, and given by different authorities as high as 80% (Gowers)[1] and 90% (Barker),[2] the discrepancy becomes evident.

This, then, is the first point, that in comparison to brain tumors or their surgical equivalents, other conditions causing papilledema are exceedingly infrequent.[3]

For the second portion of this study the hospital records of the last 100 cases of verified brain tumors showing papilledema were investigated. Of these, it was found that 48 had been treated in one or more ways for their choked disks before

[1] Gowers, Sir William, quoted by Osler. (*The Principles and Practice of Medicine.*) D. Appleton & Co., 1910.

[2] Barker, L. F. (*The Clinical Diagnosis of Internal Diseases,*) vol. 3, p. 565. D. Appleton & Co., N. Y., 1916.

[3] It may be said that one other condition, namely, nephritis, may induce changes in the fundus that simulate choked disks very closely. It was impossible to investigate the numerous renal cases at this time, so that this group has been omitted from consideration. Urinary examination being such a routine, however, it must be very seldom that a differential diagnosis is not made, and in addition there are nearly always characteristic changes in the peripheral retina.

being referred to a neurological surgeon, while the remaining 52 were referred to this clinic as soon as the papilledema was discovered. The forms of therapy employed were as varied as they were frequent, and a total of 58 procedures had been carried out on the 48 previously treated patients. Of this number there were 23 instances coming within the provinces of ophthalmology or oto-laryngology. The most frequent procedure was a change of glasses, which occurred fourteen times. This form of therapy no doubt was instituted usually by an optician, but in not a few cases an ophthalmologist had been consulted. Other frequent measures were the removal of tonsils and adenoids and operations upon the nose or paranasal sinuses. A complete view of the wide range of treatment with the figures for each type may be seen in Table II.

TABLE II.

Types of Treatment Carried Out for Alleviation of Papilledema	*Number of Instances of Each Type*
1. Change of glasses	14
2. Pituitary or thyroid extract given	5
3. Tonsils and adenoids taken out	5
4. Gastro-intestinal measures	5
5. Operations on nose and nasal sinuses	4
6. Symptomatic measures for "epilepsy"	4
7. Treatment for "neurosis"	4
8. Osteopathic treatment	3
9. Antiluetic treatment	3
10. Teeth removed	2
11. Miscellaneous (one each)	9
Total procedures	58

To illustrate more in detail what has been the course of treatment in individual patients whose choked disks were not at once assumed to be due in all probability to a brain tumor, a few specific examples may be given. The cases chosen represent both sexes, and ages varying from 17 to 48. Tumors of many kinds and in various situations are represented, such as gliomas, endotheliomas, and acoustic neuromas, so that it is obvious that no single tumor type is entirely responsible for the confusion, although in reviewing the 100 tumor cases it ap pears that the acoustic growths are

on the whole more likely to have had previous treatment on an erroneous basis.

The first case to be given was an example of the last named variety of neoplasm. This patient had consulted seventeen physicians before the cause of her varied discomforts was discovered.

CASE I.—P. B. B. H. Surg. No. 14294. *Left acoustic neuroma with advanced pressure symptoms. Vision reduced to light perception with the right eye and to* $\frac{20}{100}$ *with the left.*

April 4, 1921. Admission of Harriet R. K., 26 years of age. She had first noticed impairment of hearing in the left ear in July, 1918, and consulted an otologist. The difficulty was believed to be due to wax in the external meatus. The wax was removed without improvement in hearing. She returned to the same man two or three months later, and this time it was thought that the condition was "catarrhal." The eustachian tube was inflated and she was told that the other ear could be saved from impairment. Some weeks later the same procedure was carried out by a second aurist. No relief ensued. In Dec., 1918, after an attack of headache and vomiting, glasses were prescribed for hyperopia. She was also given a diet at this time on the basis that her vomiting had been due to a gastro-intestinal disturbance. In March, 1919, she was again examined, this time by an internist, and after a gastric analysis, rest was prescribed. During August, 1919, she began having severe headaches which finally became localized to the suboccipital region. Gastro-intestinal X-rays were taken and she was treated with belladonna without relief.

In December, 1919, she first complained of blurring vision, and in January, 1920, her eyes were again refracted. She was treated for a gastric disorder for the third time during January to March, 1920. In the summer of this same year she noticed for the first time some staggering and unsteadiness. These symptoms progressed rapidly and by January, 1921, her gait had become "drunken" in character. By this time also her vision had failed to an alarming degree, and after consulting an "oculist" she was told that she was losing her sight because of some focal infection. Her appendix consequently was removed in February, 1921, while her tonsils and adenoids were excised a month later.

Upon admission to the Brigham she presented the full-blown syndrome of a left cerebello-pontile angle tumor. Vision O.D. showed light perception only, while V.O.S. was

reduced to $\frac{20}{100}$. Her fundi showed receding papilledema with advanced secondary atrophy consequent to it.

On April 8, 1921, Dr. Cushing exposed and partially removed a left acoustic neuroma. She was discharged to her home May 3, 1921. Her pressure symptoms were relieved and her ataxia improved, but her optic nerves had been damaged beyond the point of very useful vision before her intracranial operation could be undertaken.

COMMENT.

The foregoing case was perhaps an extreme example, especially among the more recent patients, but such a story of delay was not infrequent among the earlier records of even five to ten years ago. There was no information in the history as to the earliest appearance of the papilledema, but her vision began to fail in December, 1919, and although choked disks may be present for a long time before visual impairment is noted, the reverse could hardly occur. In addition, the fundus examination at the Brigham showed that her optic nerves had long since passed through the more acute choked stage and had gone on to nearly complete secondary atrophy.

The next case was likewise an angle tumor. The record of this patient gave a definite statement concerning the onset of papilledema.

CASE 2.—P. B. B. H. Surg. No. 12722. *Left acoustic neuroma with extreme impairment of vision, marked cerebellar ataxia and general pressure symptoms.*

July 3, 1920. Admission of John C. R., 25 years of age. His illness began in Sept., 1919, with headaches, which were soon followed by vomiting and dizziness. On Oct. 14, 1919, he consulted an ophthalmologist who discovered that he had what was termed "optic neuritis." He stopped work in March, 1920, and consulted another ophthalmologist, who noted that he had "double choked disks, easily + 4 D on each side," and also noted that there were "nystagmic jerkings" to right and left. The second examiner thought of the possibility of brain tumor, but influenced, it appears, by a neurological colleague attempted to explain the fundus picture on another basis. In discussing this question he said, "It is difficult to classify these 'choked disks' with the so-called spurious optic neuritis of advanced degree. To be sure, the appearances of the choking are not exactly

those seen in the papilledema of brain tumor, but even so they seem to be greater than those of spurious neuritis."

The patient's vision continued to fail gradually until June, 1920, after which there was rapid impairment until he was admitted to the Brigham Hospital.

Examination upon entrance showed marked ataxia and incoördination, with positive Romberg's sign, persistent nystagmus to right and left, and some tenderness to pressure in the suboccipital region. It is remarkable, in view of the subsequent tumor verification, that he showed no loss of hearing in either ear at this time, either subjectively or by the usual tests. The eyegrounds showed a bilateral papilledema of 4 D, along with advanced atrophic changes and much new tissue formation. There was complete embedding of the vessels in many places. Vision was reduced to $\frac{1}{10}$ with the right eye and $\frac{1}{100}$ with the left.

On July 8, 1920, the cerebellar region was explored by the writer and no tumor disclosed, although intracranial tension was great. The pontile angles were not explored and the operation left as a decompression.

This patient improved subjectively in everything except his eyesight, showing that although his pressure had been removed, his optic nerves had already suffered beyond repair. He went along very well for three years, when his ataxic symptoms again became more marked and he returned to the Brigham on January 26, 1924. This time he had well marked deafness in the left ear and a left facial palsy, indicating an angle tumor.

On February 4, 1924, Dr. Cushing removed nearly completely a left acoustic neuroma, and his general condition has remained very good since that date.

COMMENT.

The interesting, although unfortunate feature in this case, so far as our present subject is concerned, was the attempt on the part of an ophthalmologist to explain the fundus picture on some other basis than that of tumor. A measured elevation of 4 diopters could not conceivably be caused by anything but mechanical pressure, a point which was stressed a few years ago by Dr. Cushing.[1]

The following patient was one of the three in the series who were treated on the basis that the condition was luetic. For-

[1] Cushing, Harvey. "Accessory Sinus Disease and Choked Disk." *Jour. Am. Med. Assn.*, July 24, 1920, 75, pp. 236 and 237.

tunately, the old idea of a course of mercury and iodides has gradually faded out, so that now, relatively few patients are subjected to delay on this account.

CASE 3.—P. B. B. H. Surg. No. 19941. *Gliomatous cyst of left cerebellar hemisphere. Blindness of right eye, and vision in left eye reduced to* $\frac{5}{100}$ *of normal.*

October 21, 1923. Admission of Goldie A. A., 17 years of age. The present illness began in September, 1922, about one year before her admission, with headaches, accompanied by vomiting, which occurred without relation to meals. Very soon she developed a staggering gait. Because of the increasing severity of these symptoms and the onset of pains in the back and joints, her tonsils were removed in March, 1923. After this procedure her headaches were worse, requiring morphine for their relief during a period of several weeks. In April she consulted an oculist because of failing vision and diplopia. He prescribed glasses for hyperopia, but vision continued to fail progressively. During the whole course of her illness she was given mercury and iodides daily until two weeks before she came to Boston.

Examination upon admission disclosed bilateral papilledema of 5 D with secondary atrophy well advanced. V.O.D. showed light perception only, while V.O.S. was $\frac{5}{100}$. Her cerebellar symptoms consisted in a positive Romberg, some moderate ataxia and definite hypotonia.

On November 2, 1923, both Dr. Cushing and the writer participated in an exploration of the cerebellar region. A gliomatous cyst of the left hemisphere containing 40*cc* of fluid was evacuated, and its lining treated with Zenker's solution. She was discharged on November 24, 1923, and although improved in other respects, her vision remained as it had been just previous to her operation.

COMMENT.

Similarly to Case 1, no positive date was given in this patient's record as to the time when her papilledema was first discovered, but presumably this must have been in April, 1923, six months before she was referred for operation, because at that time her vision began to fail and continued to fail progressively thereafter. Furthermore, the advanced atrophy and greatly reduced vision which were disclosed upon her admission, bespoke a process of long duration.

The following patient was also treated for lues, in this instance after choked disks had been discovered, and despite an inconclusive Wassermann reaction.

CASE 4.—P. B. B. H. Surg. No. 18952. *Glioma of left motor area. Vision of both eyes reduced practically to blindness.*

June 2, 1923. Admission of Nina E., aged 46. For about one year she had complained of headaches, which were soon followed by petit mal seizures. In October, 1922, her vision began to fail and during the next five months she consulted osteopaths and bone-setters without relief. In November, 1922, and again in March, 1923, she had convulsions with asymmetry of the face, and in the latter month she consulted an ophthalmologist, who told her that she had choked disks and had a blood Wassermann taken. The latter was reported anticomplementary, but she was given a course of K. I. nevertheless. During this treatment the ophthalmologist told her that her eyes were improving in spite of a subjective diminution in vision. In May, 1920, she had to stop work because of poor sight, at which time she consulted the physician who immediately recommended operation.

Upon admission to the Brigham she showed marked mental deterioration, dullness, and disorientation. There was a slight right facial weakness, and tenderness over the left temporo-frontal region of the skull. She was having severe headaches. Both pupils were dilated and scarcely reacted to light. She was blind in both eyes, except for bare light perception. The fundi showed bilateral choked disks of 4 to 4½ D. There were large amounts of new tissue over the disks and many hemorrhages radiating from the disk margins.

On June 11, 1923, the writer explored her left motor area and disclosed a tumor of the face center. Four days later this tumor was removed at a second-stage by Dr. Cushing. It proved histologically to be a glioma.

She was discharged June 27, 1923. At that time she was relieved of headache and her general neurological symptoms were improved greatly. Her blindness, however, remained complete, though her disks were flat.

COMMENT.

This patient proved to have a type of glioma which was quite encapsulated, and therefore offered a relatively good

prognosis after removal. Her usefulness, however, was practically at an end because she had been allowed to become blind before an intracranial operation was advised.

The following case had an encapsulated tumor which was successfully removed. Fortunately, her vision had suffered no marked impairment, but her focal symptoms were so definite that it would seem hardly possible that a correct diagnosis had not been made earlier.

CASE 5.—P. B. B. H. Surg. No. 19275. *Endothelioma of left motor area which had attained the weight of 162 grams before operative removal. Bilateral papilledema of 2–3 D.*

July 19, 1923. Admission of Clara J. T., 48 years of age. Fifteen months before entrance she began to have headaches and some mental dullness. In September, 1922 (ten months before admission), she had a convulsion with twitching of the right arm and leg, after which she limped and dragged the right toe, while the right hand felt numb and weak. Four weeks later she commenced to have vomiting at intervals. She was given the Sippey diet for a month and her vomiting ceased, but she still dragged her right foot. In February, 1923, she had two more focal convulsions, and in April another period of vomiting. She was told that the latter was a "nervous condition" incident to the menopause. A small growth on the inner side of her lip was noted at this time and was excised after another convulsion had occurred. She failed progressively with increasing weakness of the right arm and leg, and finally Dr. C. F. Hoover was called in consultation. He made a diagnosis of brain tumor and referred her to this clinic.

Examination showed a marked degree of mental dullness and beginning aphasia. There was a right partial spastic hemiplegia, with paralysis nearly complete in the arm. Her fundi showed bilateral choked disks of 3 D on the right and 2 D on the left. She could not be made to coöperate in a test for visual acuity. X-ray of the skull showed in the posterior frontal region the typical bony changes overlying an endothelioma.

On July 30, 1923, Dr. Cushing enucleated a large endothelioma from the region indicated. She made a most satisfactory recovery and was discharged August 21, 1923, with strength returning rapidly in the right side. She was able to read ordinary print at this time, but her optic disks, although flat, showed a moderate degree of secondary atrophy.

COMMENT.

Here, then, was a patient who had received a common form of symptomatic treatment in the presence not only of severe, but of localizing neurological manifestations. In her case, no oto-laryngological problem was involved, and fortunately also her eyes had not been severely damaged. The case is presented merely to illustrate that a long period of delay was allowed although the evidences of brain tumor were almost unmistakable. Had the neoplasm been one of more rapid growth it is probable that vision would have been distinctly impaired.

SUMMARY AND CONCLUSIONS.

It would appear from this investigation that the answers are clear to the questions which were proposed at the beginning. In the first place, although there are a few conditions other than brain tumor which may cause papilledema, or a condition of the optic nerves simulating it, the percentage of instances in which it occurs in these conditions is extremely small as compared to tumor. Second. The previously assumed etiology and treatment of 100 consecutive patients who had papilledema due to tumor was erroneous in 48 per cent. It cannot be stated with certainty that such treatment was instituted in all cases after swelling of the disks had begun or was disclosed by examination. In many instances, however, this was stated definitely in the record, and in others the amount of vision lost, and the appearance of the optic nerves after arrival at the clinic, evidenced a process which must have been present over a considerable period of their previous therapy. Third. The delay incident to prolonged treatment upon an erroneous assumption resulted in some damage to the optic nerves in every instance. This damage was severe in 27 patients, of whom several had reached the stage of blindness. The detailed records of five patients have been presented in order to give more specific and individual evidence of these points.

It may be concluded then, that in the vast proportion of cases, papilledema means brain tumor, or at least a surgical

intracranial condition equivalent to it. A neurologist or neurological surgeon should be given the earliest possible opportunity to see a patient in whom choked disks are discovered. Though a neurological surgeon is no more exempt than others from diagnostic error, he at least is likely to err therapeutically on the side which favors preservation of vision rather than on the side of procrastination in the vain hope that a choked disk may be due to something less serious than tumor.

I wish to express my thanks to Dr. Cushing for his permission to report these cases from his clinic.

PAPILLEDEMA IN OTO-LARYNGOLOGY.[1]

By Dr. LEON E. WHITE, Boston, Mass.

THE presence of papilledema in oto-laryngology is an inconstant, imperfectly understood and often disputed entity. Not a little controversy has arisen especially between the brain surgeons and the rhinologists as to the possibility of its ever originating from infections in the accessory sinuses, teeth or tonsils. It is hoped that from this symposium a better understanding may arise, that its importance in the diagnosis of the various intracranial infections may be more correctly stated, and that a more satisfactory explanation may be given of its presence in one case and absence in similar ones.

In brain abscess, meningitis and lateral sinus infections the otologists desire to make a correct diagnosis as early as possible and experience has demonstrated that considerable help may be obtained by frequent examinations of the eye grounds, in spite of the fact that any of these intracranial infections can occur with a normal nerve head. As Jobson well says: "Absence of ocular symptoms does not justify the exclusion of intracranial involvement, but their presence, when considered in conjunction with other symptoms, is a valuable guide to a timely operation."

The writer felt that an analysis of a large number of these cases should prove of value, so the following data has been taken from the records of the Massachusetts Charitable Eye & Ear Infirmary of the ophthalmoscopic findings in one hundred and seventy-six cases of brain abscess, meningitis and lateral sinus infections arising from the ear, nose and

[1] Read in Montreal on Sept. 17, 1924, before the Amer. Acad. Ophthal. and Oto-Laryn.

throat. These records cover a period of 30 years and represent cases covered by many surgeons. The fundi examinations were by some one of the ophthalmologists, usually the surgeon on duty, and many thanks are due Doctors Quackenboss, Spalding, Derby, Lancaster, Verhoeff, Thompson and others for their painstaking reports. The terms used by the various examiners differ somewhat; for instance, one uses the term "optic neuritis" to describe what another designates as a "blurring of the disk margins," so that while an effort is made in classifying the fundi changes to adhere as closely as possible to the words of the examiners, it was necessary to collect these reports into certain groups for study and comparison. It is frequently difficult to draw the line of demarcation between these various groups as, for instance, the blurring of the disk margins and a "commencing" or "early" papillitis. "Clinically," Eagleton says, "it is seldom possible to differentiate between an optic neuritis and a papilledema. . . . The nerve head manifestations of brain abscess, usually are a combination of both. . . . The causes giving rise to them are not thoroughly understood."

Classifications of the eye findings.

1. Marked papilledema, *i.e.*, 2 diopters or over, usually with hemorrhages.
2. Slight or early papilledema, *i.e.*, under 2 diopters, yet with some elevation of the disk.
3. Blurring of the disk margins—often designated as optic neuritis.

The above classes all showed some optic nerve changes, while the following had no change in the nerve head.

4. Dilatation of the retinal veins, *i.e.*, enough change to keep them out of normal but in the percentages they are classed as normal.
5. Normal.

BRAIN ABSCESS.

Mention of the fundus findings were found in fifty-six brain abscess cases from middle ear infection and two from nasal

infections, of which nineteen were cerebellar, thirty-seven temporal sphenoidal and two frontal.

In the nineteen cerebellar abscesses, the fundi were normal in six, slight engorgement of the veins in two, blurring of the edges of the disk in two, slight papilledema in five, marked papilledema in four. That is, eight of the nineteen cerebellar cases (or 42%) were practically normal, while 58% showed optic nerve changes. This differs considerably from the findings of Friesner and Braun, who state that "optic nerve changes were present in sixteen of the eighty-six cases," *i.e.*, 18.6% as contrasted with 58% in my series. They also state "the condition found is usually an optic neuritis but occasionally there is a choked disk." In my cases nine or 50% of all the eye changes were classed as papilledema and but two or 8% as optic neuritis. In three of my cases the changes were more marked on the affected side, in one on the opposite side and the impairment of vision was slight.

In the thirty-seven temporal sphenoidal cases the fundi were normal in fifteen, blurred edges in four, slight papilledema in five, marked papilledema in thirteen, *i.e.*, 60% showed optic nerve changes. The nerve changes were more marked on the affected side in seven cases, while in only one case was the swelling on the unaffected side the more pronounced.

My findings are quite similar to Dickie's, who states that "the condition of the eye grounds was noted in thirty-four cases and in twenty-six of them there was some change and the condition was generally more marked on the affected side." He refers to Adson who "found choked disks in nine out of his twenty-six cases."

In my thirty-seven cases of temporal sphenoidal abscesses there were twenty-six fatalities, nine of which showed marked papilledema and three blurring of disk margins. Nine were normal, *i.e.*, 65% had optic nerve changes. Of the eleven cases which recovered four had marked papilledema, one blurring of the disk margins and six were normal, *i.e.*, 45% had some optic nerve disturbance. Thus, the nerve changes were somewhat more common in the fatal cases than in those which recovered.

Of the twelve fatalities from cerebellar abscess, one had marked papilledema, three slight or commencing, two blurring

of the disk margins, and six normal, *i.e.*, 50% had optic nerve changes.

Of the seven cases which recovered, three had marked papilledema, four blurring of disk margins, *i.e.*, all the cases had some optic nerve changes, a rather interesting point when contrasted with the 50% in the fatal cases and the 45% in the temporo-sphenoidal cases.

In the two frontal abscess cases the fundi were normal although the spinal fluid was under pressure. This accords with Eagleton's findings:

"In frontal lobe abscess papilledema is rarely present. An examination of the records of over one hundred cases of frontal lobe abscess shows only three to have exhibited definite nerve head changes, all associated with symptoms of compression. The almost uniform absence of papilledema in frontal lobe abscess is due to the fact that papilledema is largely dependent upon an obstruction of the central cerebro-spinal fluid circulatory system."

MENINGITIS.

Records of the fundi changes were found in sixty cases of meningitis from middle ear infection and three from nasal infection.

The fundi were normal in the three nasal infection cases although the spinal fluid in all was under pressure and contained bacteria.

Of the sixty meningitis cases from middle ear infection, thirty-two had normal fundi, two showed slight dilatation of retinal veins, sixteen blurring of disk margins, three slight or early papilledema, seven marked papilledema, *i.e.*, 43% had optic nerve changes.

Of these sixty cases there was a record of the lumbar puncture findings in thirty-eight. The spinal fluid was under increased pressure in thirty-two while in six it was normal or subnormal. The fundi were negative in sixteen of these cases with spinal fluid under pressure, or 50%. It was normal in six and not noted in the remainder.

In the ten cases with papilledema, the spinal fluid was under pressure in seven, not noted in three.

In the two cases with slight engorgement of the retinal veins, spinal fluid was under pressure in all.

In the sixteen cases with slight blurring of disk margins, spinal fluid was under pressure in seven, normal in three and not noted in six.

In these sixty cases of meningitis there were but two recoveries. In one there was slight blurring of the disk margins, the spinal fluid was turbid, under pressure, 800 cells but no bacteria were found. In the second case the fundi were normal at first, later the "nasal borders were blurry, disk hyperemic, Hylus obscured and fine vessels unusually distinct, veins distended and large vein in right showed pulsation," the condition being noted as one of early papilledema. The spinal fluid in this case was under pressure, turbid, 65,000 cells. Culture showed large cocci in short chains, probably streptococci. A later lumbar puncture showed spinal fluid clear, not under pressure, 270 cells, no growth.

Respiration became labored while removing the spinal fluid in one case of marked papilledema and the patient passed out probably from herniation of the brain into the foramen magnum. It is always well to bear in mind this possibility when advising lumbar puncture.

The bacteriological examination was recorded in twenty-eight cases. Two were staphylococci, fifteen were streptococci, five were pneumococci, six no bacteria was found.

In the six cases with normal pressure of the spinal fluid, bacteria were noted in four, all streptococci.

In the thirty-two cases with spinal fluid under pressure, examinations for bacteria were recorded in fifteen. Streptococci were found in six, pneumococci in three, staphylococci in two, no bacteria in four.

In the ten cases with papilledema, bacteria was reported in three, all streptococci; no growth in two; no record in the remaining five.

In the sixteen cases with blurring of the disk margins, the fluid was examined for bacteria in five; in four streptococci were found; and in one no growth.

In the thirty-two normal fundi cases, spinal fluid was examined for bacteria in fourteen; four showed streptococci; two staphylococci; five pneumococci; three no growth.

The two cases with dilated retinal veins had streptococci in the spinal fluid.

It was interesting to note that streptococci were the only bacteria found in the cases with optic nerve disturbances and that in none of the seven cases where other bacteria were found were there fundi changes.

The importance of early examinations of the fundi in suspected meningitis is well emphasized by Kopetsky, who states.

"At an early stage the increase in the amount of cerebral spinal fluid produces a strangulation effect on the blood vessels of the optic nerve, and causes an eye-ground picture which is fairly constant and highly significant. It results in a swelling of the papilla, and blurring of the outer margins of the disk, which must not be confounded with nor is it the real choked disk seen later in the disease. This sign, taken in connection with what has gone before, is of great diagnostic value."

The sixteen cases with negative fundi but increased spinal fluid pressure is contrary to Beck's statement in discussing Dr. Jobson's paper on "The Ocular Symptoms of Brain Abscess and Sinus Thrombosis of Otitic Origin." He says:

"The cause of optic neuritis, or choked disk, I think has been proved to be a mechanical impediment. Dail of Prague, at the Russian Medical Congress, showed the way the veins enter the optic nerve sheath. This fact led me to a study of the relation of the spinal fluid, or rather, the result we get from spinal puncture associated with choked disk. I have never seen a case of intracranial trouble in which the spinal puncture came with a marked spurt, that did not also show a choked disk. Conversely, in the absence of the choked disk the spurt of the spinal puncture has not occurred."

LATERAL SINUS INFECTIONS.

Records of the fundi changes were found in sixty cases that had infection in the lateral sinus. Of this number there was a thrombus found in thirty-six. The remaining twenty-four bled on incision of the sinus, although in some the vein walls were thickened and a mural thrombus was noted. Twenty of the thrombus cases had normal eyes, three slight engorge-

ment of the retinal veins, five blurring of disk margins, two slight papilledema, six marked papilledema. Of the twenty cases without an obstructing thrombus, eight had normal eyes, one slight engorgement of the retinal veins, four blurring of disk margins, five slight or early papilledema, two marked papilledema.

Forty per cent. of the sinus cases showed optic nerve disturbances. This differs considerably from Tobey, who states that "Optic neuritis or choked disk may be present in a small percentage of cases (10% or 11%) due to meningeal irritation and infection, or from extension of the thrombus and consequent blocking of the petrosals, or even involvement of the cavernous sinus."

Lumbar puncture was done in eleven of these sixty cases. Fluid was cloudy in four, clear in seven, under pressure in nine, normal pressure in two. Of the nine cases of sinus thrombosis in which the spinal fluid was under pressure, fundi were normal in four, definite papillitis in three, slight blurring of disk margins in two. In the two cases with normal pressure of spinal fluid, fundi were normal.

Graham well states my views on the eye findings in lateral sinus infections when he says:

"The stasis in the veins of the (nerve) head has been recognized for some years. It is so fleeting and is so dependent in case of examination of the disk veins on the personal equation of the examiner that one should be very careful about allowing the sign to weigh in the diagnosis in doubtful cases. . . . The headache, dizziness and choked disk are the most frequent of these signs and have been more insisted on as diagnostic features on account of their comparatively early appearance. . . . An explanation of the choked disk accompanying some cases showing the above symptoms is very hard to obtain, because it does not always occur even in cases of thrombus involving the lateral sinus, the petrosals and the cavernous."

Artificial papilledema by compressing the jugular veins has always seemed to me a dubious method of diagnosis. Loeb refers to it as follows:

"O. Beck and Crow found that during compression of the internal jugular on the healthy side a definite tortuosity of the

central vein of the retina could be found on ophthalmoscopic examination in jugular or sinus thrombosis. This fact is due to the interference with the venous return as a result of the compression of the healthy jugular and to the presence of the obstruction in the sinus or jugular on the diseased side. The test is not always reliable."

ACCESSORY SINUSES.

Of the cases of optic nerve involvement studied by me, about seventy-five in number, only three have shown edema of the disk.

One (Case 5) was examined by Dr. Quackenboss who reported the disk of one eye pushed forward about 2 D, considerable exudate on and about it, the edges lost, veins large and tortuous. Vision fingers 1 foot. The other eye, while showing some blurring of the edges of the disk, had no swelling. Draining of the posterior sinuses was followed by a rapid disappearance of the edema of the disk and vision of $\frac{20}{20}$ was ultimately obtained although there remained slight pallor of the nerve. This case was operated on in 1917 and had no further disturbance with the vision up to the time of his death from pulmonary tuberculosis five years later.

The second case (Case 9) was referred by Dr. Verhoeff whose examination showed marked unilateral engorgement and swelling of the disk of about 3 D, vision $\frac{20}{200}$. This patient also had exophthalmos. There was involvement of the antrum and ethmoids. Vision rapidly returned after these sinuses were drained. This case has been seen at frequent intervals since 1918 and the vision has remained normal.

The third case, also unilateral, was referred from the Eye Clinic of the Infirmary by Drs. Reimer and Verhoeff on May 12, 1924. They found edema of the right disk of about 2 D, vision nil. As I had this paper under preparation at that time I was anxious to get all possible data. Several of the ophthalmologists confirmed the presence of edema of the disk. The loss of vision was of but four days' duration and was preceded by an acute coriza. With the exception of a deviation of the septum to the affected side with some blocking of the posterior sinuses, the nasal examination was negative.

The radiograms of teeth, accessory sinuses, sella turcica were also negative. The optic canals were normal. The vision commenced to improve before a lumbar puncture or Wassermann could be taken, which was unfortunate as far as the study of the case was concerned, as the patient refused to return for these tests.

There has been reported many cases diagnosed as papilledema, supposedly due to infection in the accessory sinuses, which have subsided after opening them. Surgeons dealing with brain tumors usually feel that there was an error in diagnosis, that the lesion was primarily in the brain and that incidentally the condition subsided following, but not dependent on, the intra-nasal surgery.

Cushing says: "Only intracranial tension can produce choked disks; I am exceedingly doubtful whether inflammation of the sinuses can cause this symptom. A retrobulbar neuritis does not produce swelling of the nerve head. . . . A choked disk is due to mechanical, not to inflammatory causes and optic neuritis is a misnomer."

Undoubtedly there have been many cases with brain lesions and an accompanying impairment of vision that have had the accessory sinuses exenterated not only without benefit but in some instances with serious or fatal results. Cushing justly criticizes as follows.

"But in any event do not let us be misguided into the belief that no harm is done to patients with threatened loss of vision due to "optic neuritis" if they have their turbinates removed and sinuses indiscriminately opened. No greater discredit could possibly come to rhinology as a specialty."

Thorough investigation would have prevented most of these useless procedures. But, on the other hand, the neural surgeons may overlook an accessory sinus infection and submit the patient to fruitless intracranial surgery, meanwhile the vision may be seriously damaged when it might have been possible to have preserved it by the elimination of infections in teeth, tonsils or sinuses. Sluder remarks apropos to this.

"Neglect of rhinologic investigation in the presence of choked disk may also be basis for criticism, as a case referred

to me eight years ago by Dr. W. H. Luedde, shows. A normal woman, 30 years old, had had bilateral choked disk with severe headache. A bilateral brain decompression had been done, without betterment. Optic atrophy followed with loss of light perception. I found a bilateral sphenoiditis, and operated both sides. The headache was relieved, and light perception returned. . . . I had had the experience of turning the patient, losing vision, over to the neurologist for investigation, and before he finished his examination, which was elaborate but negative, the patient was blind beyond help, from sphenoiditis."

Case 27 in my series became permanently blind while undergoing neurological investigation for optic neuritis.

If, as Eagleton says, papilledema may result from some change in the composition of the cerebrospinal fluid as happens from infection in a pneumatized petrous tip associated with an acute mastoiditis, it would seem within the range of possibility that infections in the posterior sinuses might likewise produce similar changes in the cerebrospinal fluid to be followed by a papilledema. I have never seen such changes which if they ever do occur, must be extremely rare.

This is as good an explanation as any I have seen, if it is a fact that the cases of papilledema reported by the following reliable rhinologists are identical with those produced by intracranial pressure from brain lesions.

Loeb reports two cases,—one, with bilateral loss of vision had a swelling of 4 D in one eye and 6 D in the other. The second case had a unilateral swelling of 3 D with vision for light perception.

Wood and Wallis report a case with defective sight for two years in the left eye, vision for hand movements and the right eye was also somewhat affected. Vision $\frac{6}{9}$, with marked papilledema. There was pus in the nose. The removal of the middle turbinates and opening of the sphenoid brought about normal vision right and nearly normal left.

Sluder mentions a case with a swelling of the disk of 6 D in one eye and 8 D in the other, arising from double sphenoiditis. He further states:

"I have had like cases referred to me by Drs. Joseph W. Charles, A. E. Ewing, John Green, Jr., William H. Luedde, W. A. Schoemaker and Meyer Wiener, all most competent ophthalmologists, in which the neurologic examinations were made by most competent neurologists. There was no instance, however, of so great swelling as 8 D. Many of these were in observation until complete recovery of the eye. . . . Ophthalmoscope showed: right, mushroom-like swelling of disk with swelling starting abruptly at margin and mounting up. Margins very much veiled, veins a little dilated and tortuous."

He continues.

"Many times I have had eye cases referred to me with the statement of the most competent ophthalmologists, that there is a lesion, usually termed choked disk, of 1 to 8 diopters, and no neurologic or other cause can be found for it. I have been able to recognize hyperplastic sphenoiditis, with often acute processes added, in these cases, and have relieved the eye trouble by the postethmoidal-sphenoidal operation."

Stucky reports "three cases of 'choked disk' due to suppuration in the sphenoid sinus or posterior ethmoid cells. . . . Nearly all of them were complicated by a systemic condition. . . . The diagnosis of the accessory sinus condition was verified by subsequent operation."

Stark in a review of the literature in 1915 found five cases with choked disk among fifty-two cases of nerve involvement from the accessory sinuses. Of these cases "one was from maxillary sinus, ethmoid and sphenoid trouble. One was posterior ethmoid. In the other three the location is not given."

Stark reports one of his cases.

"A man, aged 34, apparently healthy in every way reported to me that the vision of his right eye had materially decreased in the last few days. Examination disclosed central scotoma for color, pupil slightly larger than normal, no double vision, disk elevated 5 D, with the usual venous

stasis, otherwise, it was normal. . . . A Wassermann test was made which gave a reaction. The case cleared up in a short time under antisphilitic treatment, leaving normal vision. To my mind this must have been a case of syphilitic involvement of one of the posterior sinuses; otherwise we could not account for the improvement with suction."

From these reports by men of unquestionable skill and integrity, reports which might be duplicated many times, the fact seems well established that there are cases of edema of the optic disk which arise from accessory sinus infections.

The next and in a way the crucial point is the one of nomenclature and it is this nomenclature that is, I believe, responsible for most of the controversy.

Are these edemas from the accessory sinuses identical with those from intracranial pressure? While both have swollen disks and appear alike, would it not be possible to differentiate them? In the edemas from intracranial pressure the loss of vision is gradual or it may even be normal, while in those from the accessory sinuses it is sudden and usually complete.

In the former there are no sensory disturbances about the eye, while in the accessory sinus edemas there is usually lameness and sensitiveness. In the edemas from intracranial pressure there are generally other neurological symptoms, while in those from the accessory sinuses there are usually none. It seems probable that in the accessory sinus edemas there are inflammatory swellings with resulting exudates in and about the nerve which produce a much more serious constriction than those from infiltration of the cerebrospinal fluid. In many of the papilledemas associated with intracranial infections we have the history of a suppurative middle ear, the patient is critically ill, his system is being overwhelmed by the septicemia and the eye condition is only an incident. While in the edemas from infections in the sinuses, the loss of vision is the distressing symptom, the infection is a localized one in and about the optic canal, and the general system is but slightly disturbed. In spite of the ophthalmoscopic similarity in the appearances of the nerve heads in these dissimilar conditions, there is little question but that etiologically we are dealing with two very distinct optic nerve dis-

turbances. It would, therefore, seem infinitely wiser if we could designate them by different names. For instance, the swelling of the nerve head (which, let me interpolate, is very rare) from accessory sinuses, teeth or tonsils, being in the nature of a neuritis plus some swelling, would be well designated by the word "optic neuritis with edema," while the word "papilledema" should be reserved for the swellings associated with intracranial infections and the neoplasms accompanied with increased intracranial pressure.

The following quotations from Sluder supports these views.

"Neurologists, for the most part, think that choked disk can be produced only by intracranial pressure, and that it is edema of the disk produced by cerebrospinal fluid pressed out in the sheath of the optic nerve. Ophthalmologists recognize what is the same lesion, or one identical with it, and indistinguishable from it, that certainly arises from other causes also. Neurologists sometimes think that they are the better judges of what is choked disk, which may account for some of this difference of opinion.

. . .

"I wish to emphasize that choked disk is a lesion that seems to be of more than one etiologic mechanism; that the term choked disk describes a swelling of the nerve head and is not specific for its etiology; that the lesion secondary to postethmoidal sphenoidal lesions is in all probability an optic neuritis; and that by the ophthalmoscope it may be indistinguishable from that produced by intracranial lesions."

There is one phase of this subject that needs special emphasis, *i.e.*, loss of vision from the intracranial neoplasms in patients with definite nasal pathology. The ophthalmological examination reveals a fundus that might be due either to early intracranial pressure or to accessory sinus infection and the patient is referred to the rhinologist who finds infected sinuses or suspicious tonsils. As this might be the explanation of the eye condition there is liable to be no further attempt to investigate the case. The removal of the infection within the nose or throat naturally does not benefit the vision of patients with multiple sclerosis or acoustic neuromas and it

is in this way discredit comes to the science of rhinology. Cushing bitterly chastises us for neglecting to properly investigate these cases. Thanks to his prodding they are now being investigated much more thoroughly. It has been my good fortune in several instances to discover the more serious intracranial lesion and advise against nasal operations. The history of the onset of the amblyopia is so typical, either of its nasal or central origin, that this in itself should put us on guard. The slow progressive loss of vision, nearly always bilateral in the cases of central origin, warrants at least the necessary time for investigation. While in the sudden unilateral loss of vision cases, especially with normal optic canals, needless haste is unnecessary. Many of these will recover spontaneously. My three cases with swollen disks were unilateral and all had sudden loss of vision. If the rhinologist would restrict his surgery to these acute unilateral cases he would rarely have occasion to be censured for unnecessarily opening the accessory sinuses. While bilateral edemas of the disk from intranasal infections have been reported, they are rare and need much more careful investigation, especially if the loss of vision is gradual.

My experience within the past three years has convinced me of the advisability of letting the accessory sinuses alone unless they show evidence of infection, and to look elsewhere for the focus. My only reservation would be that in those cases with abnormally small canals (*4mm* or less) it might be necessary to ventilate the posterior sinuses as a therapeutic measure, to prevent optic atrophy. These are, however, very unusual cases.

SUMMARY.

Papilledema when present in meningitis, brain abscess and lateral sinus infection is a valuable diagnostic aid, but its absence does not exclude intracranial involvement.

The data from the Massachusetts Charitable Eye & Ear Infirmary seems of especial value as the eye examinations were made by the ophthalmologists who were not particularly interested in the findings, *i.e.*, their attitude was always neutral.

Nerve head changes are classified under three headings.

1. Marked papilledema.
2. Slight or early papilledema.
3. Blurry disk margins.

Dilatation of retinal veins being more or less of a transitory disturbance is classed with the normals.

Optic nerve changes were found in 58% of the cerebellar abscess cases and in 60% of the tempero-sphenoidal cases.

The fundi were normal both in the frontal abscess, and meningitis cases, arising from nasal infections. Forty-three per cent. of the meningitis cases from middle ear infection showed optic nerve changes.

Normal fundi were found in 50% of the meningitis cases with increased spinal fluid pressure. Streptococci were the only bacteria found in the cases showing optic nerve changes.

Forty per cent. of the infected lateral sinus cases had optic nerve changes.

The fundi were normal in 45% of the lateral sinus cases with spinal fluid under increased pressure.

Edema of the disk was found in but 4% of the optic nerve disturbances arising from infections in teeth, tonsils or accessory sinuses.

Swollen disks from accessory sinuses, teeth or tonsils is an unusual condition, is transitory and is always associated with sudden and marked impairment of vision. Papilledema is the usual condition in brain tumors and before advocating exenteration of the accessory sinuses a very careful study of the neurological side of the case should be made. In these edemas of the disk the burden of proof is on the rhinologist and I should advocate no intranasal work until the possibility of brain lesion has been eliminated.

The so-called papilledema from infections in the accessory sinuses, teeth or tonsils is not identical with that associated with brain tumors, brain abscess, meningitis and lateral sinus infections. It would seem advisable to designate the edemas associated with sinus infections as "optic neuritis with edema" and restrict the word "papilledema" to the edemas of the disk caused by increased intracranial pressure.

BIBLIOGRAPHY.

JOBSON, J. B. "Ocular Symptoms of Brain Abscess and Sinus Thrombosis of Otitic Origin." *Trans. Amer. Acad. Ophth. and Oto-Laryn.*, 1914.

EAGLETON, WELLS P. *Brain Abscess.*

FRIESNER and BRAUN. *Cerebellar Abscess.*

DICKIE, J. K. M. "A Contribution to the Study of Brain Abscess." *Trans. Amer. L. R. and O. Soc.*, 1922.

KOPETZKY, S. J. "Contribution toward the Early Diagnosis of Meningitis." *Trans. Amer. Acad. Ophthal. and Oto-Laryn.*, 1912.

TOBEY, G. L., JR. "Lateral Sinus Infection, Diagnosis, Treatment and Complications." *Trans. Amer. L. R. and O. Soc.*, 1921.

GRAHAM, H. B. "Difficulties in the Diagnosis of Sinus Thrombosis." *Trans. Amer. Med. Assn. Section L. R. and O.*, 1915.

LOEB, H. W. *Operative Surgery of the Nose, Throat and Ear*, vol. ii.

CUSHING, HARVEY. "Remarks on the Acoustic Neuromas and on Ethmoidal Operations for Choked Disk." *Trans. Amer. L. R. and O., Soc.*, 1920.

SLUDER, G. "Borderland of Rhinology, Neurology and Ophthalmology." *Trans. Amer. L. R. and O. Soc.*, 1921.

LOEB, H. W. "Two Cases of Blindness Relieved by Ethmoid Exenteration." *Trans. Amer. L. R. and O. Soc.*, 1920.

WOOD and WALLIS. "A Case of Bilateral Papilledema and Ring Scotoma Due to Sphenoidal Sinusitis." *The Lancet*, 1910, p. 937.

STUCKY, J. A. "Some Serious Eye Conditions the Result of Intranasal and Nasal Accessory Sinus Disease." *Trans. Amer. Acad. Ophthal. and Oto-Laryn.*, 1913.

STARK, H. H. "Sudden Blindness due to Suppuration of the Accessory Nasal Sinuses." *Jour. A. M. A. Section Ophthal.*, 1915.

"Retrobulbar Neuritis, Secondary to Diseases of the Nasal Sinuses." *Trans. Amer. L. R. and O. Soc.*, 1921.

WHITE, L. E. "Loss of Sight from Retrobulbar Neuritis due to Posterior Accessory Sinus Disease with Report of 17 Cases." *Trans. Amer. L. R. and Soc.*, 1919.

"Accessory Sinus Blindness, Differential Diagnosis and Operative Technic." *The Laryngoscope*, vol. xxxi., No. 8, Aug. 1921.

THE SIGNIFICANCE OF PAPILLEDEMA TO THE OPHTHALMOLOGIST.[1]

By Dr. JAMES BORDLEY, Jr., Baltimore, Md.

IN 1908 Doctor Harvey Cushing and I published a series of papers outlining some clinical observations and experiments dealing with choked disk. In these papers we expressed the view that the preponderance of evidence adduced by us strongly pointed to a confirmation of a mechanical causation. Time and additional evidence have but more firmly convinced me of the correctness of this idea.

Indeed, today I think of choked disk, or, if you will, of papilledema, only as a visible manifestation of an increase in intracranial pressure. The more firmly this view becomes fixed the more certain do I become that the treatment of choked disk does not properly belong in the domain of the ophthalmologist. It is but a component of a symptom complex that clearly indicates a situation serious enough to demand immediate consultation. As choked disk is most frequently produced by intracranial tumor growth, the ophthalmologist who delays consultation and full investigation assumes large responsibility. He is not justified in holding such a patient, even though the disk changes are slight and the vision normal. Vision is indeed a very unreliable basis upon which to predict the ultimate state of the nerve or even the duration of the choking. Choked disks may persist over a long period of time without in the slightest interfering either with the field or acuity of vision. On the

[1] Read in Montreal, Sept. 17, 1924, before Am. Acad. Ophth. and Oto-Laryn.

other hand, without apparent alteration in the choking the vision may undergo rapid and permanent deterioration. We can be sure of one thing—permanent alterations in the field and loss of acuity invariably take place if choked disks are allowed to run their course, and in a very large proportion of cases complete blindness will supervene before death terminates the story, and indeed only too often where the primary lesion will not lead to a fatality.

Any permanent alteration in the true structure of the nerve will result in a corresponding change in sight, and it is well to bear in mind that this fact is not altered by treatment. Nor should we be unmindful that the longer the process continues the greater will be the loss in vision. Therefore, the ophthalmologist should lose no time in seeking expert advice outside of his own sphere of action. This advice should be sought, no matter to what extent vision has been damaged, because as de Schweinitz and Holloway have so aptly stated: "All who have to do with the blind will realize how much it means to the sufferer if he has only light perception and how he will cling to that small blessing with almost the same tenacity with which we should cling to the preservation of good sight. Therefore, it would seem from even this standpoint it is fair to give the patient a chance."

Choked disks are not always the result of tumor growth. They may prove to be the only manifestation of some hidden intracranial disturbance which spontaneously disappears, leaving behind no evidence of the causative factor. Undoubtedly, such cases have been confused with tumors where other symptoms than papilledema were late in making their appearance. So I think it extremely wise whenever we find a swollen nerve unassociated with other symptoms to have tucked back in our minds that the intracranial tension resulting from some types of tumors is very variable, even at times permitting entire subsidence of swelling for periods of weeks and even months, and that choked disks frequently antedate other symptoms by months. Under the circumstances it is unwise, I believe, to conclude that because a choked disk has diminished in size, or disappeared entirely following some small extracranial operation, that the focal infection prompting the operation must be the source of trouble. Experienced

observers have all, I am sure, noted marked subsidence in swelling following operative shock and blood letting only to see the choking back again in a few weeks.

In the past attempts have been made to differentiate various types of choked disks. Unfortunately, there is nothing peculiar to a choked disk of any given cause to indicate that cause. We only know that when we find the papilla swollen we have unearthed a symptom of some intracranial disturbance. To the physician looking for a solution of an immediate problem this information is not very inspiring, as it has called sharply to his attention a process which may lead to blindness and even death without providing the means for an urgent diagnosis.

It is a comfort, however, to know that by a carefully executed process of elimination we can generally arrive at a fair conclusion. We know, for instance, that in simple meningitis swelling of the disk is extremely rare, except where an obstruction hydrocephalus complicates the late stages of meningitis, where it becomes almost inevitable. Its appearance is indeed a valuable indication of such a complication. Choked disk is likewise extremely rare in cerebral pyogenic abscess (doubtless because the brain is destroyed rather than pressed upon) except where there is considerable local edema, in which case there may be considerable rise in intracranial tension. This does not hold for tuberculous abscesses which follow closely the symptomatology of brain tumor. In the choked disk of nephritis we may have the typical picture of tumor, or find it associated with macular figure, depending upon whether there is a simple cerebral edema or edema complicated by extensive vascular changes in the retina. Syphilis is probably the most frequent of all "medical causes" in the production of choked disk and its presence should of course always be determined.

Foci of infection remote from the brain and optic nerve may be provocative of choked disk. Such a complication is, however, extremely unusual and such a cause while requiring investigation should not be too seriously stressed in the mind of the investigator. As an instance of focal infection I mention only in passing, nasal sinus disease. It has been my privilege to see nasal sinus disease complicated by choking of

the disks, but in every instance where the cranial contents were viewed either before or after death there existed unmistakable evidence of any intracranial complication.

I have no idea what Doctor White may have in mind to say about this phase of the subject, nor do I wish to encroach upon his preserve, but I cannot discuss choked disk from the point of view of the ophthalmologist without pointing to the prevalence of the exaggerated importance attached to sinus disease as a cause of choked disk, and the possible serious consequence upon the individual of this mistaken notion. Let me illustrate: Recently I had the opportunity ot studying two patients, one in the service of Dr. S. J. Crowe, and the other a patient operated upon by Dr. Walter Dandy, both at the Johns Hopkins Hospital. The first patient, a soldier, applied to Dr. Crowe for an operation on his right sphenoidal sinus, because he had been told it would restore his sight. The sinus had previously been operated upon and was still obviously diseased. Dr. Crowe attributed the choked disks to a probable cerebellar tumor and an immediate operation confirmed this view. One year, however, had been wasted waiting for the hoped-for result from the sinus operation, and during that time the patient had permanently lost his sight. The sinuses of Dr. Dandy's patient had also been extensively operated upon. She had a large removable cerebellar tumor, but was so devitalized by the months lost to the ravages of her malady that she barely survived the operation. Possibly in very, very rare instances sinus disease may lead to an increase in intracranial pressure through a non-purulent increase in the fluids of the intradural spaces, such as we see occasionally in disease of the mastoid process, but such a possibility must constitute a very unusual exception and should not so mislead the examiner as to make him overlook much more probable causes.

The greater my opportunities for study, the more convinced do I become that choked disk is in the great preponderance of instances dependent upon tumors of the brain. It has been my good fortune to enjoy a very exceptional opportunity in the observation of this condition, and I have so frequently seen tumors become manifest after months and even years had elapsed since the discovery of the nerve swelling

that I have grown very skeptical at snap diagnoses and remote possibilities.

Cushing and I were unable in our study of the eye-grounds to discover anything of value in determining the size or structure of a tumor. Small benign growths in some situations will, by a mechanical effect, increase the intracranial pressure more than large malignant infiltrating ones placed elsewhere. Further, as de Schweinitz has emphasized, the stage of the retinal process is no indication of the duration of the cerebral lesion, nor can we predict in a case of tumor in the absence of choked disks when this is likely to occur. The whole question resolves itself into the study of the character of the intracranial pressure and its effect on the cerebrospinal circulation, regardless of the histology, the situation, or the duration of the neoplasm. A tumor so situated as to produce an obstructive filling of the ventricles will cause a general increase in tension, often of high degree and of rapid onset. Under such circumstances, the choked disks which are practically equal on the two sides may rapidly appear. As Dr. Parker has pointed out, the intraocular pressure plays a part in determining the side which will show the first swelling. Having discussed with him his theory and having enjoyed the opportunity of seeing some of his painstaking experiments, I am convinced of the correctness of his views.

As all choked disks from serious causes run practically the same course, except where interrupted by treatment or death, it might be well to try to establish some standard of change for the determination of prognosis and treatment. Marcus Gunn attempted this by dividing the progress of the swelling into five stages. His classification of the physical changes in the disk and the result on vision of treatment seem so well authenticated by other observers that it would be folly to attempt a new arrangement. As he described it: The first stage is that of increased redness and blurring of the superior and inferior nasal disk margins; the second, beginning filling of the cup with blurring of the temporal margin and a tendency of the edema to spread into the retina; third, decided increase in edema and elevation of the nerve head associated with the forming of striæ between the disk and macula, and retinal hemorrhages; fourth, mounding and opaqueness of

the disk, disk and retinal exudations, associated with an increase in size and number of hemorrhages; fifth, decided subsidence in vascularity and increasing pallor, with or without sinking of its prominence, contraction of the retinal arteries with thickening of their perivascular lymph-sheaths and spots of degeneration in the retina.

As the damage to the nerve and retina in the first three of these stages is but slight, the vision is usually good and the prognosis as to sight is excellent if treatment of the primary lesion is successful. Where the primary cause can neither be located nor eradicated, subtemporal decompression should be resorted to during the first three stages because the dangers attending the operation are relatively few as compared with its benefits.

When the fourth and fifth stages are reached it is almost inevitable that some deterioration of sight will follow, either with or without operation. Fortunately, however, decompression frequently prevents blindness and occasionally restores some degree of vision even in these late stages. Indeed, if but a vestige of vision remains I am apt, other things being equal, to advise operation. The suggestion of the operation should at this time never carry with it a favorable prognosis because after the edema subsides the vulnerable nerve fibers are gripped and compressed by the new tissue formation in the nerve and its effects can never be calculated. Occasionally complete blindness follows close on the heels of the operation.

Knowing the disadvantages as well as the advantages of decompressive operations, the ophthalmologist may feel great hesitancy in advising operation in unchanging choked disks showing only the primary stages. He must, however, remember that tumors may continue to grow and still show a fluctuating degree of neuroretinal edema, even to complete subsidence of the process; that there may be periods of temporary relief to an obstructive hydrocephalus through escape of fluid; that spontaneous hemorrhages into the substance of a vascular tumor may cause a sudden rise in intracranial tension with a resulting elevation in the height of the disks, which subside again as the blood is absorbed; cysts which possess a variable degree of tension may develop in degenerated

areas of a tumor; and finally, an advancing growth may lead to a local cerebral edema in its immediate neighborhood with temporary increase in tension and a swelling of the disk which lessens as the edema passes away.

These variations in intracranial tension may greatly lengthen the life of the victim's sight, but the minute infiltration of the nerve is sufficient to provoke degeneration and atrophy there will be a permanent visual loss. This degree of infiltration may be long delayed, may take place gradually, or it may come with astonishing suddenness. It is my opinion, be the cause what it may, in these protracted, so-called "mild" choked disks that surgical intervention should be advised, because continued swelling, even without progressive change, is a sure indication that intracranial tension is not falling and without such fall it can reasonably be expected that vision will ultimately deteriorate. If I am correct in thinking that increased intracranial pressure is the cause of choked disk, and am secure in the knowledge that choked disk produces blindness, what other advice could I give?

I have no desire to seem radical, indeed, my chief cause for worry in the past has been my conservatism. Still when one stops to think that a large proportion of the individuals unoperated upon lose their sight, and that palliative decompressive operations offer no serious menace to sight and not much more to life, if performed by a capable surgeon, why should we hesitate? Drugs are essential in certain underlying causes, such, for instance, as syphilis; but if tried they should not be persisted in beyond the danger point of the choking. It is better to relieve the intracranial pressure first and then administer the drug. One method promises sight, the other possible blindness. Neurologists and surgeons may base their advice upon the presence or absence of other physical signs, but for the ophthalmologist the saving of vision must be the determining factor. It makes little difference to the patient whether we hold to the mechanical views of Schmidt-Rimpler, or to the toxic theory of von Leber, so long as we open for him a way to preserve his sight.

ABRUPT MONOCULAR BLINDNESS, RESULTING IN COMPLETE OPTIC ATROPHY, DURING ENCEPHALITIS WITH DOUBLE PAPILLITIS. X-RAY OF OPTIC CANALS.[1]

By Dr. CLIFFORD B. WALKER, Springfield, Mass.

(*With two illustrations on Text-Plate V.*)

CERTAIN terms like retrobulbar neuritis, rheumatism, polyglandular, etc., are sometimes used to cover a multitude of evils. As knowledge accumulates these terms characteristically become captions outside a bracket with increasing subdivisions and occasional separation of distinct entities. A feature of these subdividings is that in the first place they are often accomplished or accompanied with much argument, and in the second place, by a tendency for the observer to attempt to explain too many conditions by crowding them toward the entity which he is more clearly defining or studying.

This is particularly true in the case of certain optic neuropathies originating in the neighborhood of the bony contacts of the nerve, since it represents a region considered by three or four groups of specialists, the ophthalmologist, the rhinologist, and the neurological surgeon as well as the internist. It has not always been possible for these groups to get the same viewpoint. The controversies have always been very beneficial to all concerned, since the discussion naturally lead to an intense study of all the factors and a widening of the visual angle. It seems only a short time ago since the argument between neurologist and ophthalmologist concerning the mechanism of choked disk was active. Then later concerning the value

[1] Read before *Am. Ophth. Soc.*, Hot Springs, Va., June, 1924.

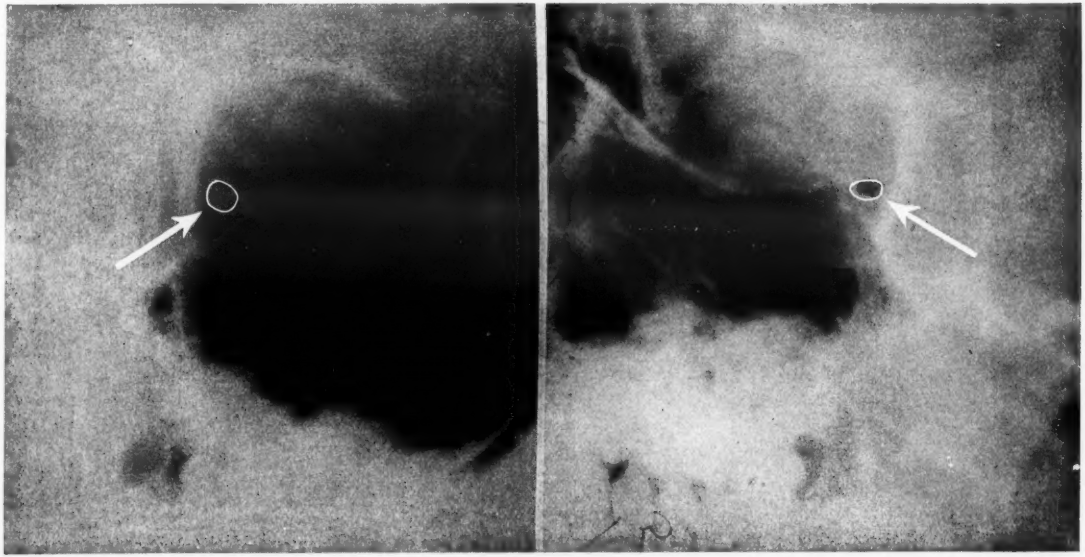
of the intranasal lacrimal sac operation between the rhinologist and ophthalmologist. Now more recently we have had some controversy and much enlightenment concerning the ability of the sinuses neighboring the optic canal to produce visual defects, and perhaps we may be about to form different opinions concerning the reliability of the X-ray in measuring the size and significance of the optic canal in the same or similar conditions.

Onodi probably laid the groundwork for the present discussion of optic canal neuropathics by showing that visual disturbance without ophthalmoscopic manifestation (retrobulbar neuritis) could be produced by post-ethmoid sphenoid infection. He also emphasized the importance of the size of the bony canal not only of the optic nerve but also of numerous other nerve canals in the neighborhood of these sinuses. His idea that these canals might be narrowed by hyperplastic process of the bone or periosteum brought on by inflammation of the sinus mucosa. In this country Sluder has given a most excellent exposition of these conditions. Shaeffer has emphasized the tendency to encroachment on the cross-section area of the optic canal when pneumatization of the ethmoid and sphenoid cells are more extensive than usual especially if the root of the lesser wing of the sphenoid is excavated.

Van der Hoeve and especially Leon White of Boston have carefully taken X-rays of the optic foramen in order to check these results in the first place, and to use the findings as a prognostic diagnostic and therapeutic guide. Dr. White's valuable contribution has interposed a further stay, in certain cases, to the hand of the rhinologist whose readiness and limited viewpoint has drawn comment from Professor Harvey Cushing and also from Alan Woods.

Still more recently Menninger has reported choked disk and optic neuritis and loss of vision in the course of influenza and has traced reports of similar instances, reported in the literature back for thirty-five years or more. Hiram Wood also exhaustively surveyed this subject and encephalitis lethargica subsequent to the epidemic of 1918. Perhaps the most recent report that of Parmenter and Cheney cites a case of encephalitis lethargica with double papilledema of 2 D. elevation but no disturbance of visual acuity. It seems well to keep the fore-

ILLUSTRATING DR. C. B. WALKER'S ARTICLE ON "ABRUPT MONOCULAR BLINDNESS, RESULTING IN COMPLETE OPTIC ATROPHY, DURING ENCEPHALITIS WITH DOUBLE PAPILLITIS. X-RAY OF OPTIC CANALS."



Right Optic Canal

Left Optic Canal

going facts and consideration in mind in studying the following case.

Admission October 1, 1923, No. 1655, Wesson Hospital, D. K., age 10, white, male.

P. H. Usual diseases of childhood, measles, whooping cough, mumps. Tonsil and adenoids removed at age of 7.

P. I. Four or five days previous to present illness, patient was in swimming on water wings at Water Shop Pond. He went under when the wings collapsed and was rescued by the attendant. The onset began with headache and malaise followed by nausea and vomiting but very slight temperature so that for a week or so it was treated as a gastro intestinal upset. But in the second week when seen by Dr. H. D. Davidson, to whose kindness I am indebted for the privilege of making this report, the symptoms were more meningeal, the headache became severe and prostrating. The back and neck became stiff and painful and a tendency to opisthotonos developed. Kernig and Babinski were suggestive at the end of the week. I was asked to see him because vision in the left eye was noted to be failing rapidly during the day. By the end of the day when I arrived, he had a large central scotoma and could barely count fingers. A mild papilledema elevation 1 D. of the optic nerve could be made out on the right side and more marked condition of the same sort on the left. There was no nasal discharge.

On admission patient complained of burning spine, sore neck and frontal headache. Patient vomits everything except water about 2 teaspoonfuls at a time.

P. E. No abnormal discharge from eye, ear, nose or throat. All mucosa normal in appearance except coated tongue. Palate reflex normal. Patellar reflex normal. Babinski and Kernig suggestive, superficial abdominal reflexes. No enlargement lymphatic glands.

October 2, 1923.

V.O.D. $\frac{4}{10}$ direct pupillary reaction normal. Consensual from left eye nil.

Fundus O.D. Papilledema with measurable elevation of 2 D. Marked engorgement and tortuosity of vessels. No hemorrhages or exudates.

Field Colors O.K. possibly some contraction, no scotoma. Only a rough test could be made because of extreme pain and opisthotonos.

V.O.S. nil. Pupillary reaction nil, consensual from right eye normal.

Fundus O.S. Papilledema 2 D. elevation. Picture same as in O.D. but slightly more aggravated.

Temp. 100. Pulse 84. Resp. 26. Very restless, severe pain in back and head, nourishment refused. Difficulty in swallowing.

Lumbar puncture under ether, gave a yellowish fluid slightly turbid, (slight contamination with blood) pressure 20*cmm* cell count of 31 per *cmm* 80% lymphocytes, bacteriological examination negative. Improvement began immediately following lumbar and continued without interruption until patient left hospital (fifteen days later).

X-ray Findings were negative as far as the sinuses were concerned but other features will be described later.

October 7, 1923.

V.O.D. $\frac{4}{4}\frac{8}{8}$.

Fundus O. D. disk elevated 1½ D. and now seems slightly worse in appearance than O.S.

Field normal to such tests as can be made with ordinary disks. Doubtless an enlarged blind spot could be demonstrated by refined methods.

V.O.S. nil. Disk still elevated 1½D. but has improved slightly in appearance.

October 11, 1923.

Temp. normal and patient is slowly improving, taking nourishment increasingly and sleeping well free from pain, but papillitis does not improve as rapidly as general condition of patient.

October 17, 1923. Discharged improved.

V.O.D. $\frac{4}{4}\frac{8}{8}$. V.O.S. nil, elevation of disk still about 1D. No evidence of secondary changes marked.

November 20, 1923. Vision unchanged. Right optic disk practically normal. Left optic disk shows the pallor of atrophy with slight secondary changes. The recovery during the convalescent period was slow.

The subsequent recovery period was uneventful except for the rapid blanching of the optic nerve. There was at no time the slightest perception of light or pupillary reaction in the left eye.

X-RAYS, DIAGNOSIS, AND TREATMENT.

We would have liked to have found evidence to regard this case as an infection primarily of sinus origin with a possible dehiscence or very thin spot in the left canal wall, thus giving us more possibility of improving vision by operation or sinus treatment. However, even if it could be considered a sinus infection either primarily or coincidentally, the question of operation in the presence of an encephalitis, with temperature

and possible meningitis, would indeed be serious. Then again the history of the case, with onset of headache, malaise, nausea and vomiting continuing to outspoken encephalitis with meningeal irritation and double papillitis of 2D. elevation followed by improvement after lumbar puncture and eventual complete recovery except for left optic atrophy corresponds more closely with numerous reports of influenza encephalitis than with the sinus group in which the measurable elevation in the disk is against the rule, as Alan Wood has carefully pointed out, and as I have found to be true in a previous case report. Now while we may diagnose this case as encephalitis probably of influenza origin we are still left without a good explanation of the following question.

Why, in the presence of practically the same degree of optic neuritis and disk elevation in both eyes, should one eye abruptly lose all vision and proceed rapidly to optic atrophy, while the other eye showed practically no visual defect throughout?

The valuable work of Leon White on the size of the optic canal as studied by X-ray suggested to us that there might be a difference in the size of the two optic canals in our case, by the same method described by Dr. A. S. Macmillan of the Massachusetts Charitable Eye and Ear Infirmary, Boston,—that is the nose, malar bone, and jaw touch the plate, and the forehead is a finger breadth away, while the ray is directed through the parietal protuberance of the opposite side and perpendicular to the plate. In this clinic it was found that in 25 cases of optic neuropathics (largely sinus cases) the X-rays showed that a difference of at least of ½*mm* or more occurred in 9 cases as between the two eyes of the same individual.

Not being able to find reports of canal measurements by X-ray on cases of the character herein described we obtained the X-ray shown in the accompanying illustration which shows a round canal on the right of 5*mm* while the left or blind eye shows a flattened canal of 4 x 5*mm* after allowing for the ½*mm* enlargement. Dr. White's work indicates that 5*mm* is the critical measurement below which visual defects are more apt to occur when the proper conditions exist to produce a swelling of the nerve, and it seems quite possible that such an explanation may account for the rapid blindness and atrophy in this

case, although it may be some time before enough cases of this type are collected to supply conclusive evidence.

COMMENT.

It would seem perfectly possible to use either the large or small size of dental film held by a strip of adhesive tape over the lower temporal quadrant, since the outline of the canal must appear in this position to be of any value. It is often necessary to take several films, especially the first cases, and if films of the sinuses are not necessary or have already been sufficiently studied, waste of film will be saved. Since the picture of the canal must appear close to one of the fixed points (zygomatic process) the error which may result from slight warping of the film will be trivial.

It is to be hoped that others will be able to find some value in this method of adding to our information of this none too completely understood mechanism of visual failure. And further that some operation even in the presence of influenza or encephalitis may be devised to either decompress the canal or lessen its rigidity sufficiently to conserve eyesight. Possibly something more at least than ventilation of the sinuses will eventually be attempted.

CONCLUSIONS.

1. X-rays of optic canals to determine size and shape may prove of value in prognosis and treatment not only in neighboring sinus disturbance but also in other conditions in which a swelling of the optic nerve may take place, as for instance in influenza encephalitis.

2. Dental films may be used if necessary for this purpose with practically no error.

3. Lumbar puncture is one of the most efficient procedures to produce relief and improvement in the encephalitis group.

BIBLIOGRAPHY.

MENNINGER, KARL A. "Optic Neuritis and Choked Disk in Influenza." *Arch. of Neurolog. and Psych.*, 1924, Vol. 2, pp. 328–333.
(This article contains an extensive bibliography from a neurological and medical standpoint.)

WHITE, LEON E. "An Anatomic and X-Ray Study of the Optic Canal in Cases of Optic Nerve Involvement" March, 1924, *Annals of Otology, Rhinology and Laryngology.*
(This article contains results of previous papers as well as new work and bibliography.)

WOODS, HIRAM. "Lethargic Encephalitis." *Trans. Amer. Ophth. Soc.*, 1919, p. 306.
(Extensive review of literature from ophthalmological standpoint.)

WOODS, ALAN, and DUNN, J. R. "Etiologic Study of a Series of Optic Neuropathies." *Jour. A. M. A.*, 1923, Vol. 80, pp. 1113–1117.

VAN DER HOEVE. "Optic Nerve and Accessory Sinuses." *Annals Otol., Rhin. and Laryngology*, June, 1922.
(Also *Arch. of Ophth.*, Vol. 51, No. 3.)

ONODI, A. "Optic Nerve and Accessory Cavities of the Nose." *Ann. of Otol., Rhin. and Laryng.*, 1908, Vol. 27, p. 132.
(Also "Der Schnerv und die Nebenholen der Nase," Vienna, A. Holder, 1907.)

SCHAEFFER, J. P. Monograph on the Nose and Olfactory Organs.

ROBERTS, W. H. "Blindness of Obscure Nasal Origin." *Arch. of Ophth.*, Vol. 53, No. 1, p. 19.

PARMENTER and CHENEY. "Encephalitis Lethargica with Choked Disk." *Boston Med. and Surg. Jour.*, Vol. 190, No. 22, p. 928.

WALKER, C. B. "Value of Quantitative Perimetry in the Study of Post-ethmoidal Sphenoidal Sinusitis Causing Visual Defects." *Boston Med. and Surg. Jour.*, Vol. 185, No. 11, p. 321.

SERUM AND LENS-ANTIGEN EXTRACT TREATMENT FOR THE PREVENTION AND CURE OF CATARACT.[1]

By Dr. A. EDWARD DAVIS, New York.

TWO years ago the author read a preliminary report on this subject before an International Congress of Ophthalmologists at Washington, D. C., and gave the results obtained by this method of treatment in thirteen patients with twenty-two cataracts. Since then fifty-nine additional patients with 109 cataracts have completed the treatment, making a total of 72 patients with 131 cataracts. In the present paper further observations and the progress of the original 13 cases are brought up to date, together with the results obtained in the present series of patients.[2]

It is only within comparatively recent years that vaccines and serums have been used clinically to any great extent for the prevention and cure of diseases. It was not until 1889 when Buchner, and later, 1890, Behring and Kitosato, presented their investigations on immune sera that an active interest was taken in this branch of therapeutics. Of these remedies, diphtheria antitoxin is the best known—although many bacteriolysins, cytolysins, opsonins, etc., either as active vaccines or passive serums, have been used with a greater or lesser degree of success.

In the present investigations both specific lens sera and lens-antigen extract were used to arrest the progress of incipient cataracts and to absorb mature senile cataracts. Perhaps a

[1] Read before the Am. Ophth. Soc., Hot Springs, Va., June, 1924.

[2] For detailed report of the original thirteen cases see *Trans. of An. International Congress of Ophth.*, held at Washington, D. C., April, 1922.

word as to the nature of cytolysins is in place here. Cytolysic sera are produced by injecting tissue cells (antigen) of one species, *e.g.*, red blood-cells, nerve tissue, crystalline lens, etc., into the blood of another species. A lytic substance (amboceptor or antibody) is thus formed, which is called a *hemolysin* when red blood-cells are used, a cytotoxin or cytolysin when other tissue cells are used as the antigen. Most of the cytolysins are *species* specific; that is, they react only on the species from which the tissue or antigen is obtained. A few of the cytolysins are *organ* or tissue specific to a greater or lesser degree, *e.g.*, spermatozoa, the testicle, the ovary and of the tissues of the eye, the crystalline lens and the uveal pigment. We are indebted to Uhlenbuth for the discovery of the organ specificity of the lens.

Present Experiments.—The lens-antigen extract and serums for the present experiments, begun February 1, 1921, were made for me by Dr. Russell L. Cecil, of New York City.[1] The first experiments were concerned with the effect of intravenous injections of anti-lens serum into patients suffering with cataract. The anti-lens serum was prepared in three different ways. 1. By immunizing rabbits against normal guinea pig lenses (anti-guinea-pig-lens rabbit serum). 2. By immunizing rabbits against human cataract lenses (anti-human-cataract-lens rabbit serum). 3. By immunizing a sheep against normal beef lenses (anti-beef-lens-sheep serum). In view of the great similarity in chemical compositions of cataract lens to normal lens, there seemed to be no reason for confining the study to the effect of anti-cataract-lens serum. The comparative scarcity of cataracts would have made such a study impracticable; hence the use of the sera from animals immunized with normal guinea-pig or beef lenses. Later on the unpleasant effects of the serum led to a study of the effects of lens-antigen when injected directly into patients with cataracts. This also appeared more rational on theoretic grounds, as more lens antibody was probably made available by active immunization than by attempts to confer a passive immunity with serum.

---

[1] Since August, 1922, H. K. Mulford Co., of Philadelphia, have furnished both sera and lens-antigen extract.

THE RATIONALE OF SERUM AND LENS-ANTIGEN EXTRACT TREATMENT OF CATARACT.

It is necessary first to consider briefly the etiology or causes of cataract, and second the nutrition of the crystalline lens, to understand the underlying principle involved in the serum and lens-antigen extract treatment of cataract. J. Burden-Cooper in his masterful Doyne Memorial Lecture[1] (1922) on the "Etiology of Cataract," states that "Subcapsular senile cataract is undoubtedly a chemical decomposition of the lentoproteid, and the trend of opinion now is that it is more acceptable to regard it as due to alterations in the whole organism than to any mechanical explanation based on local causes, and more especially a still unproven pathological contraction of the lento-nucleus." I think we may go even a step further and adopt Roemer's theory, "The sub-capsular senile cataract is just as truly a metabolic disease of the lens as, for example, diabetic cataract." In other words, he regards it as a *specific metabolic disease* and that the spontaneous development of a cataract is due to a direct toxic action upon the cells and fibers of the lens." Kirkpatrick's theory of disturbed function of the endocrine system as a cause of cataract fits into Roemer's theory. I am prepared to accept Roemer's theory in the main and to lay aside as of minor importance all theories based on mechanical or local causes,—such as Becker's irregular sclerosis of the nucleus"; Schoen's "errors in and strain upon the accommodation"; Peter's "disturbance of osmosis"; Michel's "arterio-sclerotic changes in the blood vessels," etc.

To add to the perplexity of the situation; first, the exact nature of the toxins or cytotoxins that are supposed to cause cataract are not known; so we are left, so to speak, to fight an unknown enemy, in the dark; second, our lack of knowledge, as Burden-Cooper puts it, of the nutrition of the lens. "Science so far has left unsolved the problem of the nutrition of the individual cell, and the lens, owing to its complexity, presents problems as regards its nutrition of well-nigh insuperable difficulty."

I wish to call attention especially to the part that leukocytes probably play in the nutrition of the lens by supplying

[1] *British Jour. Ophthal.*, Vol. vi., 1922, p. 384.

trephones. It must be remembered that the lens is made up from epithelial cells, which develop into lens fibers, and that these cells grow and multiply from the time of birth of the individual until his death, be he ever so old, unless the lens become cataractous, when they cease to grow and the lens become a dead inert mass in the living eye. Hence, the necessity of supplying in the first place nutrition as trephones; in the second place, stimulation to growth (hormones) to the lens cells, if we are to prevent cataract. Leukocytes play a very important rôle in supplying the nutrition which can be assimilated by the lens plasma (perhaps by specific affinity) while the endrocine glands furnish in the main the hormones. To cite Carrel further[1]: "Leukocytes which, like embryonic cells, contain growth-promoting substances, have the power to supply connective and epithelial tissues with the principles necessary for growth. The food material used by lymphocytes in the manufacture of the essentials which determine the multiplication of fibroblasts or epithelial cells comes from blood serum. Thus lymphocytes and macrophages can be considered as mobile unicellular glands which transform some of the serum constituents into trephones and set them free in blood plasma or interstitial lymph as a food for fixed cells. They play the rôle of trephocytes, or nursing cells, for epithelium and connective tissue. As trephones are also present in the aqueous extracts of glandular and other tissues, it is permissible to suppose that endrocine glands, like leukocytes, may secrete nutrient substances for certain tissues, and not only hormones. Our experiments indicate that lymphocytes remain through life as a store of embryonic growth-promoting substances or trephones, which may cause a resumption of cell activity when it is needed. They are evidently endowed with a double function, destroying foreign substances and necrotic material, and promoting cell proliferation."

Now, if we accept Roemer's theory as to the cause of cataract—to toxins in the blood from faulty metabolism,—we can understand first, how the injection into the body of lens-antigen extract, which stimulates the body cells to the formation

[1] *Journ. A. M. A.*, January 26, 1924, p. 255.

of specific antibodies, which, in turn, neutralize these toxins of faulty metabolism, can retard or prevent the formation of cataract. Second, these specific antibodies formed by the injection of lens antigen, act selectively on the decomposed opaque lens fibers (that is cataract) and cause their liquefaction and absorption, thus bringing about a *cure* of the cataract.

Keeping in mind the necessity of the proper nutrition of the lens cells and fibres, and the great rôle that leukocytes and lymphocytes play in carrying the proper food (trephones) to such cells, the *rationale* of local irritation of the eye itself,—by using drops in the eye, as glycerine, dionin, calcic-alkaline-iodide ointment, etc.; or by injecting an irritating substance under the conjunctiva, thus inciting local hyperæmia, and drawing leukocytes to the eye—may also be understood. In fact, a number of ophthalmic surgeons claim that by the use of local irritants alone, incipient cataracts in the earliest stages may be arrested and vision restored to normal. Among these may be mentioned Badal, Dor, Taylor and Colonel Henry Smith.

As regards local hyperæmia, in my preliminary paper on this subject I made the following comment. "A possible explanation of why cytolytic sera do not affect the lenses of adult animals or man powerfully, may be due to the lack of alexin or complement in the aqueous humor of the anterior chamber of the eye. This curious fact is called attention to by Zinsser[1] who cites the experiments of Metchnikoff. In this fluid no alexin is present under normal conditions, but if puncture is practised and the fluid again taken after a period of two or three hours, alexin is now found, probably, according to Metchnikoff's school, because of the coincident entrance of leukocytes into this space. It is conceivable, however, that the aqueous humor may be free from alexin for other reasons than the absence of leukocytes; and an injury followed by the invasion of leukocytes is pretty sure to be followed also by the entrance of fluid elements of the blood, *i.e.*, alexin.

This fact would suggest one practical point in the attempt to absorb or liquefy cataracts in the human animal, that is, during the period that the antigen is being given, the eyeball

[1] Zinsser, *Infection and Resistance*, Second edition, p. 170.

should be massaged, or subconjunctival injections of some slightly irritating fluids be given. In fact, any safe means employed that would induce an increased flow of the plasma and along with it leukocytes into the anterior chamber of the eye; because it is absolutely necessary to have complement or alexin present in conjunction with the antibodies of the sensitized serum in order to obtain a lytic effect—that is, absorption or liquefaction of the lens.[1]

The *fundamental distinction* between the two methods of treatment—*specific lens-antigen injections* and *local hyperæmic* method—is this: The lens-antigen serves both to *prevent* and to *cure* cataract while *induced local hyperæmia* is of value only in *preventing* cataract, through increased nutrition.

The explanation is as follows: First, the lens antigen stimulates the body cells to form *specific antibodies*, which neutralize the toxines of faulty metabolism and thus *prevent* the formation of cataract; second, the same specific antibodies liquefy and absorb the opaque disintegrated lens fibers (cataract) and thus act as a *cure*. On the other hand, local hyperæmia prevents cataract through increased nutrition.

METHOD OF USE.

(1) Preparation of the patient; first, the patient's vision should be tested without glasses, then with his refraction corrected and both recorded; second the pupils should then be dilated with homatropin and a diagram of the lens opacities made, stating at the same time if the cataract is the ordinary subcapsular, nuclear or zonular, etc. A drop of eserin should be instilled into the eyes afterwards to contract the pupils and avoid the danger of inciting glaucoma.

(2) A sensitization test to the reaction of the lens protein (in this case the lens-antigen) should be given. This consists

---

[1] Bearing on this same subject the reader is referred to the "Investigations concerning the biological behavior of the blood serum toward the lens-albumen in cataract," by P. Roemer and H. Gebb (*Archiv für Augenheilk.*, 1914, vol. lxxvi., lxxvii., lxxviii.) and also a paper by Boldingo, "Anaphylactic investigations in order to ascertain if heterologous serum passes from the blood serum into the anterior chamber," *Archiv. di Ottalmologia*, 1920, vol. xxvii., p. 77.

in injecting 4 or 5 drops of the lens-antigen *intracutaneously* into the forearm of the patient, under strict antiseptic precautions—that is, the spot at the site of the injection should first be cleaned with alcohol, and after the injection again wiped with alcohol. An ordinary sterile hyperdermic syringe (preferably glass) which has been boiled may be used for the test. Hypersensitiveness to the lens protein is manifested by redness and a slight swelling at the site of the injection in 2 to 20 hours. If the reaction is very marked the following therapeutic injections of the lens-antigen should be given cautiously and in small doses, until the patient becomes desensitized, when the usual doses can be given with safety.

(3) The following day after the sensitization test, the therapeutic injections of the lens-antigen are begun. These should be given with a 10*cc* glass syringe, 21 gage needle, both thoroughly boiled, and usually the upper arm is chosen as the site of the injection because of its convenience, though the leg, abdomen or buttocks may be used, if the patient desires. *Always* the site of the injection is first painted with iodine, the injection given under the skin, then the iodine wiped off with alcohol. The first dose as a rule is $\frac{1}{4}$*cc*; in strong patients it may be 1*cc*. In women or feeble patients, the dose is increased $\frac{1}{4}$*cc* each time, or 1*cc* at a time in vigorous patients.

(4) The injections should be given every other day, or three times a week, gradually increasing the dose, as indicated above, $\frac{1}{4}$*cc* to 1*cc* at a time, until the maximum dose is reached; in women about 8*cc*; in men about 10*cc*. Exceptionally this dose may be considerably increased. The symptoms which indicate that a full or maximum dose is being reached are: shortly after the injections, a few moments, the patient complains of a slight dizziness or restlessness, or slight pain in the back or stomach—at times with nausea. The dose should then be reduced 1 to 2*cc* and held at that for a week or ten days, when it can again be gradually increased to toleration. The patient should always remain in the waiting room 5 or 10 minutes after each injection. Fifty doses constitute a course of treatment, extending over a period of about 4 months. Occasionally it may be extended to 75 doses. In patients who come from a distance and cannot spend so much time, the injections may be given every day; and in the patients who have

received this intensive treatment, they have done as well or better than those treated every other day. In fact, with further observation, it is recommended that the injections be given every day, when it is possible.

(5) During the course of injections, the patient is placed on small doses of potassium iodide, 10 to 15 drops of the saturated solution in a full glass of water at meal time. If the potassium iodide is not tolerated, the syrup of hydriodic acid, one teaspoonful in a little water, one half hour before meals is given. Also during the course of treatment, the patient is instructed to have as nearly a salt free diet as is possible. All patients are treated ambulantly, and allowed to use their eyes moderately at their ordinary vocations. In feeble, or run-down patients, general tonics are desirable.

(6) The lens-antigen extract when not is use should be kept in a cool place in an ice box, but not on the ice. Just before use, especially when the larger doses are reached, the solution should be slightly warmed—the chill taken off.

(7) Local reactions at the site of the injections are considerable at times, especially toward the close of the treatments; the arm becomes red, swollen, with much itching; the second arm, or leg, or other site may be chosen, if the reaction is too great. These local reactions, while disagreeable, are not undesirable.

(8) In patients where the treatments have been interrupted for as long as two weeks, the sensitization test should be repeated to decide if the patient has become hypersensitive to the lens protein, otherwise marked anaphylactic changes might be produced with harm to the patient. If not hypersensitive, the injections should be resumed but with the smallest dose, ½ to 1*cc*, and gradually increasing as in the beginning of treatment.

The results obtained in 131 cataracts following the serum and antigen treatment are shown in Table No. 1. The progress of the cataracts was arrested and the vision improved in 86 eyes, 65.64%; the progress was arrested without improvement in vision in 36 eyes, 27.48%. Arrest of cataract was obtained, therefore, in these two sets of cases, in 122 eyes, or 93.12%. In 9 eyes the cataracts advanced and the vision grew worse, or 6.87%. I submit this is a fair showing even

when compared with the operative treatment of mature senile cataract. The average percent. of success in the operative treatment of cataract by the average operator is not more than 90%. Furthermore, in unsuccessful cases, following operative treatment the sight is usually irretrievably lost, which is not the case in the non-operative method of treatment. Again, the non-operative treatment has the advantage of application in the incipiency of cataract when the patient has useful vision, which is retained in 93% of the cases, as shown above; while in the operative method the patient is doomed to wait for months or years until he is almost, or totally blind, before the operation can be performed with the best promise of success. While it is true that this period of idleness, apprehension and loss of occupation may be shortened to some extent by the intracapsular method of extraction (Smith and Barraquer), however, so far, except in the hands of the very expert operators, it has proved risky, the vitreous often being lost at the time of operation, and the ultimate result proving disastrous.

DURABILITY OF RESULTS.

Very properly it will be asked, and is asked by patients, how long will the good results last? Of course, time alone can give the final answer to this question. The second patient ever to receive this treatment, February 1, 1921, had been operated upon by me for a mature senile cataract in the right eye one year previously. In the left lens were a few spiculæ of opacity of the cortex at the extreme periphery of the lens, but with V. = $\frac{20}{15}$. The cataract has been arrested and the same vision $\frac{20}{15}$ has been maintained to date or for $3\frac{1}{2}$ years. A drawing of this lens made by Miss Ferret showing the few spiculæ present at the close of the treatment is a true representation of the condition to-day. And I may say that all other patients in which the progress of the cataracts was arrested and the vision improved, with *very* rare exceptions, have maintained their gain as it was at the close of treatment.

In the series of private patients, I have had the pleasure of observing the changes in the lens with the slit-lamp. It is of interest to note the clearing of the diffuse cloudiness in the lens, also to watch the gradual disappearance in many cases of the disintegrated lens fibers. In this instrument we have a

precise objective method of observing the lens changes, and do not have to rely wholly on the subjective tests of vision to determine if the patient is improving. It must be admitted, however, that improved vision (assuming that cataract is the primary cause of the poor vision) is the crucial test in these cases. Furthermore, the patient can appreciate his improvement in vision, and that is what he cares about. Such expressions as "I can see to thread a needle, which I could not do before," "I can read fine print, which I could not do before taking treatment" tell the true story after all, since restoration of function is the object sought after.

CONCLUSIONS

By the use of Lens-Antigen alone: 1. Almost every case of cataract in its earliest incipiency can be arrested, even though spiculæ or riders may not be absorbed. 2. Immature cataracts, up to 50% of loss of function, can be arrested in over 90% of the cases; and in over 65% of the cases, the vision improved. 3. In mature, or ripe cataract, the greater mass of the cataract (cortical layers) can be absorbed, but it requires a prolonged course of treatment, and besides, the nucleus is not absorbed. For this reason it is advised that mature senile cataracts should be removed by operation. 4. The necessity for *very early* treatment, that is, in the earliest incipiency of cataract, and up to that stage of cataract where not more than one half of the vision is lost, is strongly indicated from the results reported in this paper. 5. It is highly important that the qualified ophthalmologists should open their own eyes and visualize the possibilities of the non-operative treatment of cataract—its *prevention* with the *retention* of vision—and not abandon the patient to total or almost total blindness before they even attempt to do anything for him.

With Colonel Henry Smith, I am of the opinion that—"The question of cataract opens up a wide field for the physiologist. I hope that physiologists will devote more attention to the nutrition of the lens and vitreous than they have done in the past."[1] I am of the further opinion that the study of bio-

[1] Cited by Ernest Thomson in an abstract in the *British Journal of Ophthalmology*, of Smith's latest paper on this subject.

chemistry and the subject of general nutrition will go far towards the solution of this difficult problem we have on our hands—*The Prevention of Cataract.*

RESULTS.—Table No. 1.

Patients	72			
Cataract	131			
Arrested and Improved	86	equals	65.64%	93.12%
Arrested without Improvement	36	"	27.48%	
Worse	9	"	6.87%	

The case reports appear in the paper published in the *Transactions* of the American Ophthalmological Society, 1924.

ANGIOMATOSIS OF THE RETINA.

By Dr. HUNTER W. SCARLETT, Philadelphia.

(With one illustration on Text-Plate VI.)

ACCORDING to Coates any form of blood vessel tumor in the retina is rare.

Angiomatosis of the retina, called Von Hippel's disease, was first accurately described by this author (1) in 1903, although Fuchs in 1882, Darier in 1890, Wood in 1892, and Collins in 1894 described conditions which in those days were confused with arterio-venous aneurysm and detached retina with massive exudation.

Sixteen years after the onset of the disease he described, Von Hippel was able to make an anatomical study of the case which formed the basis of his original description, the eye having been removed because of absolute glaucoma. From this study, together with that of six other eyes out of twenty-four cases thus far reported, he concluded that the condition was essentially an angiomatosis of the retina, with general disease of the retinal vessels, destruction of nervous elements, proliferation of glia, sub-retinal bleeding and organization of exudates.

In 1912 Von Hippel (2), discussing this condition, reviewed his original case. There was an extensive detachment of the retina down and out, while to the temporal side of the disk there was a large red mass lining the interior of the globe, and consisting of coils of capillary blood vessels, supported by neuroglial tissue. The large main vessels seemed to run through this angiomatous structure. Similar masses were present more anteriorly, in the inner layers of the retina, and projected into the vitreous. In the sub-choroidal space as well as in the retina near the disk, were numerous hemorrhages.

The vessels showed endarteritis, endophlebitis, and thrombosis. The central vessels appeared practically normal. In several places on the surface of the choroid, cicatricial tissue and bone were present. In one spot a retinal vessel was found communicating with a choroidal vessel. Both were enormously enlarged. Other pathological changes present were those of iritis and secondary glaucoma.

As stated above Von Hippel believed the process was fundamentally a formation of vascular tumors in the retina, the enlargement of the vessels being secondary to this tumor formation; while exudation, hemorrhage, and detachment of the retina were secondary to the vascular condition. The similarity in color between the arteries and veins was probably due to the potency of communication between them through the enlarged capillaries. He thought this condition was distinctly different from that described by Coates as "Retinal Disease with Massive Exudation," although the underlying cause of the exudates might have been the same.

Ophthalmoscopic diagnosis was comparatively easy. The characteristic appearances included one or more huge round reddish tumors, with which one or more greatly enlarged tortuous arteries and veins communicated. Retinal vessels were diseased, retina became detached, while hemorrhage and exudates were present throughout the fundus. The disease was chronic, prognosis bad, vision was gradually lost, and the eye went on to a state of absolute glaucoma, requiring enucleation.

In Frenkel's (3) study of twenty cases, one reported by himself and nineteen others collected from the literature, fifteen were in males and five in females. Seven cases were bilateral. The ages at which the condition usually appeared ranged from ten to thirty-three years. Moore (4) saw a woman aged twenty who first noticed impairment of vision three weeks after an attack of quinsy. When she first came under observation the condition was bilateral and had existed for two weeks. In a patient under surveillance for seven weeks, with detachment of the retina, Frank Kamensky (5) observed the detachment increase, tumors develop, and the vision decline from counting fingers at three-quarters of a meter to light perception.

ILLUSTRATING DR. HUNTER W. SCARLETT'S ARTICLE, "ANGIOMATOSIS OF THE RETINA."

Angiomatosis of the Retina

Stern's (6) case was twenty-one years old, in whom visual troubles had existed since fourteen years of age. The ophthalmoscopic findings were given in detail, and showed the characteristic large red tumors, into which dilated vessels emptied. There were also other dilated and tortuous vessels with exudate. This case resembled the one published by Fuchs called arterio-venous aneurysm.

Bane (7), Emmanuel (8), Wada (9), Vessius (10), and Meller (11), each studied cases resembling in appearance that of angiomatosis of the retina. Emmanuel's patient was two years old. The eye had been removed for suspected glioma and examination revealed a detached retina, exudate, hemorrhage, cyst formation in the retina, with colloid and hyaline degeneration. In the inner layer of the retina there was a tremendous accumulation of large vessels, while the ciliary region showed a tumor-like formation of capillary vessels. The condition was probably congenital.

Meller, in discussing his case, said the thickening of the retina was due to a non-inflammatory proliferation of glial tissue which was primary, while alterations in the blood vessels were secondary. This view was opposed to that of Von Hippel who thought that the proliferation of glia was secondary to the vascular changes.

In 1890 Darier (12) described as arterio-venous aneurysm, a condition which he later in 1913 called angiomatosis of the retina, following the description of this disease by Von Hippel, and the reporting of similar cases by other writers. His patient at ten years of age complained of floating opacities in the left eye, and of seeing colored halos around a light. At nineteen, he began having frequent epistaxis, violent headaches, diminution of vision with the loss of the inferonasal field, and finally complete loss of vision. At twenty-three, a fixed yellowish globular mass was observed just behind the pupil. On the surface of this mass were seen retinal vessels and several red spots. Diagnosis of sarcoma was made and the eye was enucleated. Microscopic examination showed a cystic degeneration of the retina, with an increase of large vessels.

At twenty-seven, the right eye became similarly affected with floating specks, diminution of vision, and the appear-

ance of colored rings encircling objects. Ophthalmoscopic examination showed below a red mass which was taken at first for an aneurysm, then a tubercle, but little by little it assumed a grayish white appearance with a red center into which dilated vessels seemed to empty. Gradually the retina became infiltrated, torn and detached. Finally blindness ensued. Thus a case thought at first to be an aneurysm, then a tubercle, was finally diagnosed angiomatosis of the retina.

Microscopically there was noted a proliferation of the intima, thickening of the adventitia, and in places a disappearance of the muscle fibers, which explained the dilatations and the impossibility of distinguishing vein from artery. The round red spots first called aneurysms were determined, after careful examination, to be conglomerations of very fine vessels with supporting gliomatous tissue.

According to Darier, angiomatosis of the retina is rare, essentially chronic, and is remarkable for the dilatation and tortuosity of the retinal vessels. Arteries become affected first and then the veins. The dilatation is distributed throughout the course of the vessels, which in certain places become constricted and in other parts are beaded with round or ampulliform dilatations like balloons, into which veins and arteries empty. These latter formations are always at the periphery of the retina and for a long time pass unperceived.

The tissues comprising the retinal elements cause first a thickening, then a detachment of the retina, the eye being finally lost. There may develop after a long indolent evolution, either a glaucoma, a chalk-like cataract, or a calcareous degeneration in the choroid. The small brilliant spots are due to fat cells. The large whitish yellow spots, which develop later, are due to edema of the retina, following a hemorrhage.

Case Report.

Through the courtesy of Dr. Le Fever the following notes are reported under date of 2/14/22.

Mrs. I. F. thinks the vision in her right eye has never been as good as that in the left eye, but she has noticed a fail-

ure in the right eye during the last six weeks. Mild frontal headaches. Patient of nervous temperament but general health good. Vision (with correction) O.D. $\frac{2}{20}$. O.S. $\frac{20}{20}$. Refraction under cycloplegia equals O.D. $\frac{2}{20}$ (?) and O.S. $\frac{20}{20}$.

The pupils are *3mm*, equal, and react normally to light and accommodation. The muscle balance is good, showing an exophoria of 1, and a right hyperphoria of 1. The convergence angle and motility are normal.

Ophthalmoscope.—O.D. Media clear, cup closed, disk obscured except at the temporal side, center; retinal vessels full. There is an enormous vein arising from the lower temporal quadrant, about at the site of the vena and not seen beyond that, which appears to be an anomalous course of the vena, passing directly toward the nerve head where it disappears, apparently joining the central vein of the retina. It is slightly tortuous and of metallic luster. It receives several veins on its course across the retina. A second vein one third as large, runs parallel to it. At the site of origin there is a brilliant red area, apparently in the choroid, twice as large as the nerve head with gray, hazy border, and about 6 D. of elevation. The red mass appears to be beyond the retina.

On *November 22nd, 1923*, Mrs. F., age 26, consulted the writer because of a severe frontal headache over the right eye, lasting three days. Patient said the vision in this eye had been poorer than that of the left eye, but she first noticed a decided failing of vision about a year and a half previously, just after she became pregnant. Her sight has been getting gradually worse until the present.

Patient said she could not read for more than two or three minutes without developing a severe headache, lacrimation and bloodshot eye on the right side.

At four years of age, patient had an attack of diphtheria. Immediately following this, the left eye was noticed to be somewhat smaller than the right.

External Examination.—Slight enophthalmos of the left eye, with narrowing of the palpebral fissure on that side, and a slight inequality of the pupils, the left being smaller than the right. Ocular movements full, and the eyes stationary under cover. With the phorometer and Maddox rod, an exophoria of 1 for distance, and 14 for near. No hyperphoria present. Prism adduction 6. Vision O.D. $\frac{20}{100}$. O.S. $\frac{20}{20}$.

Ophthalmoscopic Examination.—O.D. Numerous stringy vitreous opacities. Disk poorly defined, but of good color. Physiological cup obliterated. The inferior temporal vein enormously enlarged to about twice its usual size extending downward from the disk slightly toward the nasal side

then turning toward the temporal side, approaching the inferior temporal artery which was also greatly enlarged. At about midway between the disk and the periphery it turned downward again, and was lost in a large, irregularly round, well defined, bright red mass, situated at the extreme periphery of the fundus just within the inferior temporal quadrant. To the nasal side of the vessels, before they disappeared within the mass, were several small whitish areas of exudate.

The inferior temporal artery, shortly after leaving the disk, became slightly larger than normal and constricted at regular intervals giving the appearance of a sausage shaped vessel. The superior veins were also enlarged and somewhat tortuous. Above the superior nasal vein was a small flame shaped hemorrhage. There was an extensive detachment of the retina in the infero-temporal quadrant extending from six o'clock to about nine o'clock. Right disk seen with -4, and the red tumor-like mass seen with $+2$D lens.

O.S. Media clear. Disks round, well defined, normal in appearance. Fundus entirely negative, except for a slight fullness of the veins.

Fields showed a decided general contraction of the right eye, with a marked cutting in the upper nasal quadrant to about 15 degrees. This corresponded to the position of the retinal detachment down and out. Field taken 2/23/22 by Dr. Le Fever showed the same defect, only not so extensive.

Tension in each eye was 15 with Schiotz tonometer.

1/9/24. Ophthalmoscopic finding of the right eye showed the disk to be more blurred and the entire fundus more obscure. The main features of the condition were the same except for an increase in the exudate, with a perivasculitis along a branch coming off from the largest vessel above described. This latter condition was not present at the last examination.

5/22/24. O.D.V. C.F. at 2 feet. O.S.V. $\frac{11}{11}$. Ophthalmoscope O.D. Numerous vitreous opacities. Disk obscure in outline and hyperæmic in color. All vessels leaving or coming to the disk, enormously enlarged so that it was difficult at times to distinguish artery from vein. Inferior temporal vein very much the same in appearance as the description given above. The corresponding artery however was increased in size and approached that of the vein. It still retained its sausage-shaped appearance near the disk. Both vessels disappeared under a large, well-defined reddish tumor-like mass about $1\frac{1}{2}$ times the size of

the disk, situated in the extreme lower periphery a little to the temporal side.

Considerable exudate in sheet-like fashion was spread out on either side of the inferior vessels commencing at a point about halfway to the periphery and extending up to the site of the red mass. To the nasal side of these vessels were smaller areas of exudate less distinct, but with entire absence of pigment.

Along the course of a branch of the inferior temporal vein was a marked peri-vasculitis, which became more pronounced as it approached the periphery, where the vessels ran over a detachment of the retina down and in.

The superior temporal and nasal vessels were likewise enlarged, but not to the same extent as the inferior ones just described. They were slightly lobulated in places and tortuous, but did not disappear under any red tumor-like masses.

The detachment noted at the former examination appeared to have increased, extending further to the nasal side.

COMMENT.

Little is known of the etiology of this rare condition, although trauma is thought to be the cause in some reported cases, while tuberculosis is considered the underlying factor in others. The fact that it has been observed at an early age in two or three members of the same family, suggests the possibility of a congenital defect. Intracranial cysts were associated in four cases of angiomatosis of the retina, reported by Seidel.

In a well-advanced case there is little difficulty in differential diagnosis. It is only in the beginning before the characteristic tumor formation has occurred, that one is unable to properly classify such a condition. At this time it must be distinguished from choroiditis, or retinitis with massive exudate. Later when detachment of the retina is present, there is usually a corresponding enlargement of the vessels, and if one searches carefully, a suggestion at least, of a red tumor can be observed.

Darier says that epistaxis, cephalgia and dazzling before the eyes are premonitory symptoms. In the case here reported no history of nosebleed could be elicited, but the patient was subject to severe and frequent headaches, with floating specks in front of the eyes.

The condition may be far advanced and still have normal central vision. On the other hand vision may diminish comparatively rapidly as in this case, where it fell from $\frac{20}{100}$ to counting fingers, within the space of about six months. The fields may present a central or peripheral scotoma, or a marked irregular sector-like contraction.

The process is usually binocular in the end, although some cases have remained monocular. Males and females are equally susceptible.

CONCLUSIONS.

1. Angiomatosis of the retina affects arteries and veins alike, but is more pronounced in the latter.
2. Tumor formation, red in color, single or multiple, is pathognomonic of this condition, and is an early development although it may pass unperceived for a long time.
3. The disease is steadily progressive with an accompanying increase of exudate and detachment of the retina.
4. Secondary glaucoma usually ensues, with complete loss of vision, requiring enucleation for the relief of pain.

REFERENCES.

1. Von Hippel. "Uber eine sehr seltene Erkrankung der Netzhaut. Klinische Beobachtungen." *Arch. f. Ophthal.*, Bd. lix., 1, 1904.
2. Von Hippel. *Graefe's Archiv f. Ophthalmologie*, 79, ii., p. 350, 1912.
3. Frenkel. "Capillary Angina of the Retina." *Annal d'Ocul.*, cxlvii., p. 161.
4. Moore. "Bilateral Angioma of the Retina." *Ophthal. Soc., United Kingdom*, xxxii., p. 76.
5. Frank-Kamensky. "Von Hippel's Dis. of the Retina." *Ophthal. Rev.*, xxxii., p. 27.
6. Stern. *Centralblat. f. Prakt. Augenheilkunde*, v. 37, p. 298, 1913.
7. Bane. *Annals of Ophthalmology*, v. 22, p. 399, 1913.
8. Emmanuel. *Graefe's Arch. f. Ophthal.*, v. 90, p. 344.
9. Wada. *Ophthalmology*, v. 12, p. 204 and 760.
10. Vossius. *Abhandlungen aus dem Gebiele Augenheilkunde.* (Marhold, Halle, 1913.)
11. Meller. *American Encycl. of Ophthal.*, vol. xiv., p. 11,192.
12. Darier. *La Clinique Ophtalmologique*, 1913–19, N. S. 5, p. 679.

PRONOUNCED TRAUMATIC DISLOCATION OF EYEBALL WITHOUT DEMONSTRABLE FRACTURE OF THE ORBIT OR RUPTURE OF THE SCLERA—REPORT OF A CASE.

By Dr. S. P. OAST, New York, N. Y.

(*With three illustrations on Text-Plate VII.*)

L. V. L., Aged 63, on October 9th while pulling down old shed, slipped and fell on back and while in this position was struck on right side of face by end of large piece of timber causing a pronounced contusion of orbital structures, and an ugly wound of the deep lower lid margin, which bled profusely. He was seen within a few minutes of the accident by his family doctor who applied a bandage and sent him to the hospital where he was seen by me about one hour after injury.

Examination.—Revealed an "L"-shaped, jagged laceration of the deep tissues of the cheek down to the bone and extending through the entire thickness of the lower lid just external to the lacrymal punctum. A marked contusion and swelling of the lids, conjunctiva and orbital tissues were present which at first led to the erroneous impression that the globe had been eviscerated and it was only by exploration with the tip of the little finger that the globe could be felt at the extreme apex of the orbit and as well as could be made out by this method, seemed unruptured and of normal tension. All attempts to expose the globe by separating the tissues were futile as the swelling of the parts in advance of the eye was too great to permit of it.

Exploration of the wound of the cheek and the orbit at this time did not disclose any underlying fracture and there was no bleeding from the nose.

No attempt was made at this time to repair any of the orbital structures because of the obscuration caused by contusion, however, the lid margin and cheek wound were sutured and a moist boric acid dressing applied.

There was no infection and at the end of two weeks the swelling of the parts had practically subsided, the palpebral fissure was closed and the lids sunken, giving the outward aspect of anophthalmia. On retraction of the upper lid and the somewhat chemotic conjunctiva, the clear cornea could be made out, was directed up and out and approximately on a plane ¾ inch posterior to the cornea of the fellow eye. The slightest motility of the globe could not be made out, being fixed in the position indicated by the tilt of the cornea. An attempt to measure this recession with the Hertel exophthalmometer was unsuccessful because of the extreme degree of sinking; the cornea was found to be beyond the range of the instrument.

The pupil was 5*mm*, irregular in outline and gave no reaction, the iris being bound down by dense annular posterior synechia. Lens clear in pupillary area, faint red fundus reflex—no details. T. to fingers normal, not practicable with instrument. Vision—counts fingers 3 feet.

The probability of fracture of the orbital walls had suggested itself and an X-ray of the head was taken but neither the antero-posterior or lateral views disclosed any suggestion of fracture. Clinical examination of nose was likewise negative for evidences of fracture.

On October 23rd (two weeks after injury) the orbit was explored under ether with a view toward discovering and freeing the globe of adhesions which held it in its receded position. Examination revealed a complete divulsion of the oblique muscles and check ligaments of the recti,—the orbital walls seemed to be everywhere intact and there were no well defined adhesions except at the margin of the optic foramen where the globe was so firmly adherent that all efforts to separate them failed. Two squint hooks were employed for traction, one placed on either side and what the operator considered the extent of justifiable force was employed without any appreciable effect. The situation of the adherent band or bands seemed to correspond so closely to the size and situation of the optic nerve that it was deemed inadvisable to sever them with scissors and blunt dissection was of no avail, so the operation accomplished nothing toward altering the original condition.

There was very little reaction following this attempt and the eye has remained quiet ever since, the only noteworthy change in the condition during the five months that have elapsed is a clearing of the media, allowing some details of the fundus to be seen, though the position of the eye and the deposits on the anterior lens capsule preclude a satisfactory examination.

The only positive ophthalmoscopic finding of moment

ILLUSTRATING DR. S. P. OAST'S ARTICLE, "PRONOUNCED TRAUMATIC DISLOCATION OF EYEBALL WITHOUT DEMONSTRABLE FRACTURE OF THE ORBIT OR RUPTURE OF THE SCLERA."

Fig. 1. Fig. 2. Fig. 3.

is a definite pallor and obscure outline of the nerve head and perivasculitis. There are no areas of choroidal atrophy which might suggest a ruptured choroid though but only a limited extent of the periphery can be examined.

The case is believed to be very unusual and is noteworthy in that there is neither demonstrable rupture of the globe nor fracture of the orbit.

The explanation for the dense adhesions between the posterior pole and the annulus of Zinn is that a laceration of these parts took place during the injury and because of the divulsion of all the structure holding the eye forward and swelling of the parts anteriorly, the eye remained in contact and permitted adhesions to form and become organized.

The other explanation I venture with considerable hesitancy and that is an incarceration of the optic nerve in the optic foramen, the orbital part of the nerve being protruded through the foramen by the blow, the sheath becoming torn and adherent within the foramen. This view is supported by the appearance of the nerve, suggesting a post-neuritic atrophy.

The case is not reported as one of traumatic enophthalmos, because in the latter condition, the absorption of orbital fat is largely held responsible for the eye's sinking. In the case here reported, there is a marked absorption of orbital fat but it is believed that here it plays a very minor role and cannot possibly account in any adequate degree for the extreme enophthalmos.

The accompanying photographs were taken March 28th, over five months after injury.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

An ordinary meeting of the Section took place on November 14th, under the chairmanship of the President of the Section, Sir Arnold Lawson.

Cases.

Mr. Basil Lang showed three cases. One was an implantation cyst, which he brought for the purpose of comparison with a recently shown case of double implantation cyst. The second patient showed what seemed like congenital pigmentation in the deep retina or in the superficial choroid. The third case was that of a man who had a blow over an eye, which subsequently went blind. He thought it probably was a syphilitic choroido-retinitis.

Dr. Rosa Ford exhibited father and daughter with congenital ankyloblepharon. The father, now aged 34, was operated upon at four years of age, and now felt no disability. The child had the external canthi slit last July, and was now able to open her eyes much better, and did not have to raise the lids so much when making the effort.

Miss Mann showed on the epidiascope micro-photographs of the human eye in various stages of intra-uterine life. The condition in these two patients was probably due to the fusion of lids, which usually terminated at the 7th month, having persisted. The father showed absence of the levator palpebræ superioris. The child probably had the same abnormality, but was difficult to examine.

Mr. H. A. LEVY showed two cases of macular disease. In the first, in a boy, it was observed from the beginning, the first complaint being of a little failure of vision. In three months it gradually developed until the whole macula was surrounded by pigmentary disturbance. The other case was in a girl aged 18, who, in the last month, had developed a patch on the macula of the left eye, and he did not know the nature of it, as Wassermann, von Pirquet and other reactions were negative.

The Relation Between Cavernous Atrophy and Glaucoma

Mr. RANSOM PICKARD read a paper on this subject, in which he contended that cavernous atrophy was a distinct entity. Its association, he said, with glaucoma was merely casual, not causal. It might exist without glaucoma and present symptoms somewhat resembling the latter disease, or it might co-exist with it, and one might influence the course and appearance of the other. He founded his thesis on seven cases, which he described in detail. None of the cases had nerve symptoms, apart from the condition of the optic nerves. The disks were pale where the lamina cribrosa was exposed, but not elsewhere, and the vessels were not contracted. The question arose as to whether these cases were clinical entities, and whether they tended to develop general nerve symptoms if the patients lived long enough. They differed from cases of ultimate optic atrophy, as there was an absence of the dead-white color, the vessels were either not contracted or only slightly so, and there were large disks and deep cups. In none of the 7 cases was the tension over 28*mm* of mercury, in most it was well below this. Cases seen for long periods showed no change at all; they seemed to arrive at a certain stage, and not to progress thereafter.

Mr. HEPBURN said he would fear to leave doubtful cases without recommending operation.

Mr. G. H. POOLEY said he went over a large number of charts of cases of chronic glaucoma and other cases closely resembling the condition, which he had seen in the last number of years. The chief omission in the paper was that of histories of progress of the cases, for the chief difference was

as to whether the cases continued to advance, and if so, whether they could be arrested by the ordinary treatment for glaucoma. One case he saw showed no alteration in the fields in four years.

Mr. LESLIE PATON spoke of a case of the condition Mr. Pickard described which had been under his own observation 15 years, and before that was under Mr. Priestley Smith, who, long before the work of Schnabel, pointed out that it was not a case of glaucoma. The man was a myope of 15 D. The first field Mr. Paton took of him was limited to the 10° circle, and there had been no change in it in all the years since. In another probable case, also, there had been no progress whatever. He did not regard the condition as common.

Mr. PICKARD replied emphasizing again the fact that after a certain stage was reached no further progress took place. It was difficult to forecast whether a case would be of short or long duration. Mr. William Lang's case made no progress in 14 years. He had seen cases of the kind which had been operated upon, and the eye went downhill. There was a similar condition in the other eye. He asked that special attention should be given to apparent cases of glaucoma but in which there was no tension.

Miners' Nystagmus: Its Diagnosis and Origin.

Dr. FREELAND FERGUS (of Glasgow) read a paper on this subject. He said that in ordinary cases of the condition its diagnosis was not difficult. The oscillatory movements of the eyeball were not always present, but there were frontal and occipital headache, tremors of the head, vascular disturbances, photophobia, vertigo, and usually a history of the onset of visual disturbances. This, he thought, suggested a wide involvement of nerve centers. Photophobia was sometimes absent, but visual acuity was practically always reduced, and this latter was of considerable value in cases in which the diagnosis was doubtful. One such doubtful case was that of a miner in a district which showed little nystagmus. This man was certified, some years ago, as having miner's nystagmus by both the examining surgeon and the referee

who examined him on appeal. Recently the diagnosis was questioned, and then it was found that the man's condition had altered considerably: he was using a stick for getting about, and was suffering from disturbance of innervation of some of the leg muscles. There were neither head tremors nor photophobia, but the patellar reflexes were exaggerated and ankle clonus was absent. No oscillatory movements of the eyes were evident on examination, and the positive range of convergence for the median position was excellent; but on extreme upward fixation there was definite diplopia. The pupils reacted regularly, though somewhat sluggishly, to light; there was no ptosis. There was no serious error of refraction, nor could he find tremulous movements in either eye, and their fundi were healthy. In each eye the visual acuteness was practically $\frac{6}{6}$. There might, he said, be nystagmus, with diminished visual acuteness without the case being one of miners' nystagmus. Still, given a mine worker with nystagmus and diminished vision, the evidence of the presence of other causes of nystagmus must be satisfactory before a negative conclusion as regards miners' nystagmus was arrived at.

Visual acuteness in miners' nystagmus might be due to one of two causes, or to a combination of them. The first was, that the rapid movements of the eyes prevented the proper stimulation of the maculæ. In the second place, it must be remembered that there was, in almost all cases of the disease, considerable pain at the back of the head, and this might indicate a lesion at the pole of the occipital cortex, involving the centres concerned with visual acuteness.

In one of his cases the man had excellent vision, and a certifying surgeon diagnosed the case as miners' nystagmus. With that the speaker could not agree. Only the left eye was said to be affected, and the man had been away from work four months. Diplopia was permanently present, due to an affection of the left eye, the vision of which was $\frac{6}{9}$ (Snellen). The man had been operated upon for ethmoidal disease. There was no photophobia, no head tremors, and the man had not seen lights appear to revolve. In certain parts of the upper field of fixation there were to be seen those jerky movements which had received the name "pseudonystagmus."

Dr. FERGUS did not consider that miners' nystagmus had yet been satisfactorily explained. It did not seem to be due to either posture or defective illumination; nor to the depth of the pit in which the men worked. He had seen cases from pits which were comparatively shallow. Mr. ROBSON, who had conducted an admirable statistical research, attributed it to working in certain soft kinds of coal. Still, cases of it did occur in workers on hard and bright coal.

A considerable number of cerebral centers seemed to be involved in the disease, and those centers were probably co-ordinated to each other. But the causal agent did not seem to affect any centers in the medulla or cord. He had thought it might be due to a definite and specific micro-organism, and this might explain cases of the condition being more abundant in some districts than in others. In support of the idea was the fact that miners' nystagmus was accompanied by a good deal of constitutional, and even mental, disturbance. He was not aware of any **post mortem** examinations having been made on cases of the disease. If such, with bacteriological examinations, were made, much light might be thrown on the subject. The blood and cerebro-spinal fluid might be examined in living subjects. The Ministry of Health, he suggested, might arrange for a competent set of men to make a careful enquiry into the matter, including an investigation of the sanitary condition of the pits where it was prevalent.

THE PRESIDENT said it had been admitted in discussions that defective illumination was a main cause of miners' nystagmus, and it had been confidently stated that in mines where the lighting was improved the amount of nystagmus had diminished. With regard to diminution of sight in the disease, it was generally recognized that there was a physiological central scotoma in most of these cases.

Dr. ROBSON (of Penarth, S. Wales) entered into a detailed consideration of the subject, and said he thought most medical men practising in the vicinity of coalfields would incline to the idea that the disease was a central rather than local one. In some coal areas in Scotland there was no miners' nystagmus; conversely, in some mines there were particular seams which were responsible for all the cases of the disease

in the mine; yet all in the mine were working under the same conditions as to temperature, depth and pressure. It seemed to be a matter of the environment rather than a personal idiosyncrasy of the worker. There was no nystagmus in American coal mines, nor in those of South Africa. In New South Wales nystagmus raged especially in pits where the coal was liable to spontaneous combustion; it became quite hot if left for only the time between two shifts. There was no proof that there had been a steady reduction of nystagmus following the adoption of the electric head-light. He considered that miners' nystagmus was a question of some derangement of the central neurons of the brain. He believed there was no nystagmus in America because the pits were shallow; some were not more than 500 feet in depth, and there were huge ventilation plants, with, sometimes, 200,000 cubic feet of air passing in at a time. In South Wales there were deep pits, and there was not an excessive amount of ventilation.

Mr. G. H. POOLEY (Sheffield) contended that the number of miners claiming compensation was no criterion of the number of cases of miners' nystagmus. In order to arrive at dependable statistics, every man working in a mine should be examined. The movements of the eyeballs in miners' nystagmus were definitely undulatory, not a slow movement in one direction and then a quick return, as in labyrinthine nystagmus. In workers in coal mines wounds of the cornea were more likely to be infective than in the case of steel workers. He was not greatly inclined towards the cerebral idea of this disease. He spoke of the cases now seen in which prolonged abstention from work produced a pitiable nervous condition in the men, of a severity which was not seen before the payment of compensation became the rule. Before that time, men often returned to their work within three months.

Mr. HARRISON BUTLER said many cases of miners' nystagmus had no definite diminution of vision when errors of refraction were corrected and the test-types were placed low down.

Mr. R. J. COULTER agreed with Mr. Butler.

Mr. BERNARD CRIDLAND could not agree with the last two speakers. He pointed out that miners' nystagmus was

practically absent in candle-lighted mines, but present in those lighted by lamps, and if a candle would not burn in a mine, a man working in such must suffer in health.

Mr. J. W. THOMAS drew special attention to the physical characteristics of the coal face. Soft coal was of a uniform dull blackness, and therefore reflected little light, a factor which should be taken into account in trying to account for this condition. The electric cap-lamps illuminated the coal a hundred times more than did the ordinary lamps used underground.

Dr. Fergus replied.

A meeting of the Section was held on December 12th, under the chairmanship of the President, SIR ARNOLD LAWSON. It was a clinical meeting, and took place at the Royal Westminster Ophthalmic Hospital.

Post-operative Result of Conical Cornea.

Mr. ELMORE BREWERTON showed a patient who came to him three years ago with conical corneæ, the right eye being the worse. He did the crucial incision through the apex of the cone of one eye (he proposed to operate on the other eye shortly). Vision in it, with correction, was $\frac{6}{24}$ ($\frac{6}{18}$ partly), whereas in the other eye it was $\frac{6}{6}$. If the apex was below the center of the cornea, it was well, he said, to go obliquely, *i.e.*, down and in. If it coincided with the center of the cornea, the incision was made horizontally. If the aqueous escaped, the second and third incisions must be made with small scissors with blunt points. The flaps were lifted up separately, and the blunt-pointed scissors passed by the side of the repositor. No tissue was removed. As a rule, the anterior chamber re-formed in five days. It was essential to have the iris fully dilated under atropine.

Bilateral Uveo-parotitis

Mr. VINER showed a patient with chronic irido-cyclitis associated with considerable enlargement of glands. He believed oral sepsis was responsible for both conditions.

Lower Half of Field of Vision Lost in one Eye

Mr. McMULLEN showed a patient with this defect and said that when he saw her six weeks after she first noticed loss of sight there could be found nothing to account for it, except that he observed a little opacity of the retina above the macula, and that the disk was somewhat pale. When taking the fields he found complete loss of the lower half of the field of the right eye. A physician found that the patient had mitral stenosis, hence it was likely that there had been an embolism of the upper main branch of the central artery of the retina.

Recurrent Retinal Hemorrhages.

Mr. McMULLEN also showed a patient who was subject to recurrent retinal hemorrhages, for which no cause could be found. These sub-retinal and retinal hemorrhages had been spreading further into the retina, with progressive failure of sight.

Gradual Disappearance of Cysts of Iris.

Sir RICHARD CRUISE exhibited a patient who had had two large cysts of the iris. Two years ago, when he showed it to the Section, there was much discussion as to what ought to be done. They had, however, been slowly getting smaller since, and now were quite small.

Modified Motais Operation.

Sir RICHARD CRUISE also showed a patient on whom he had performed a modified Motais operation. He was a big man with very heavy eyelids and congenital ptosis. The superior rectus was functioning, and the speaker split it, and dissected back a broad brim from the central portion of the muscle, leaving only the margins attached to the eyeball. He dissected that back ¼ to ½ an inch, giving a good long thong of muscle. It was firmly secured in the muscle by a double-threaded suture. He then split the tarsal plate at the posterior margin, and brought the thong of muscle through the

tarsal plate, and sutured it there firmly. There was now excellent functioning of the pseudo-levator palpebræ. The man now had nearly full movement, and was able to walk about and see comfortably.

Sir WILLIAM LISTER said he would hesitate to do the operation for fear a foreign body might get between upper lid and globe.

Mr. TREACHER COLLINS said this operation was only suitable for cases of ptosis in which the superior rectus was active. The cases in which he had done it had been unilateral ones. He did not go through the tarsus, but through the retro-tarsal fold, then burrowed with a strabismus hook between skin and tarsus and planted a piece of rectus muscle, which was sutured into that burrow, and the sutures were brought up on the skin near the palpebral margin, and a little piece of rubber tube tied in. He was much helped in the operation by using half-curved needles.

Marginal Dystrophy of the Cornea.

Mr. FRANK THOMAS (Swansea) showed two patients with marginal dystrophy of the cornea. One of them showed the ultimate condition found in this disorder, namely, marked peripheral ectasia of the upper half of the periphery, leaving the central area distorted, and by the displacement forward, a high degree of myopia developed. Vision in the right eye was $\frac{6}{18}$, in the left $\frac{6}{36}$ with correction. Neither of these patients suffered from discomfort, except for the loss of vision, which in the worst case extended over 10 years, in the other two years. Both men had suffered from blood-shot eyes.

Mr. TREACHER COLLINS agreed with the diagnosis, though the appearances in the two patients were, at the present time, strikingly different. He had examined both these cases with the slit-lamp. The man with ectasia showed the ribbon of light in the cornea definitely incurved on the posterior surface, indicating a thinning of the cornea in that region. The man with the opacity showed thinning at the anterior surface, or what might be termed a slight grooving. Ectasia caused considerable astigmatism. The case recorded by J. H. Fisher was treated by excision of a thin piece of cornea

at the margin, and the result was most satisfactory, though it seemed a hazardous proceeding. He, the speaker, did not advise excision of a piece here.

Papillœdema in a Myope.

Mr. A. Levy showed a patient whom he saw four years ago with 8 D. myopia in each eye. Vision at that date was about $\frac{6}{9}$. The man came again three weeks ago, still a myope, with vision $\frac{6}{36}$, and a flaring optic neuritis in both eyes, and the disk swelling — 1. It had been stated, he said, that a myope never got papilloedema.

Cases of Leber's Atrophy.

Miss IDA C. MANN showed, for Mr. Leslie Paton, a patient with Leber's atrophy, whose four brothers had also been shown before the Section with the same condition. There were in the family 6 brothers and 4 sisters. None of the women were so affected. The oldest brother was affected at the age of 28, the next at 33, the next at 27, the next at 22, and the present patient at 43. A brother, now aged 34, was not yet affected. Four sons were among the offspring, but none yet affected. In reply to a question by Mr. Frank Thomas, she said the skiagram of the skull showed no definite enlargement of the sella turcica.

Case of Pituitary Tumor.

Mr. MONTAGUE HINE showed a man with this condition. There was seen to be a definite enlargement of the sella turcica. He had been under thyroid treatment two months, and during that time vision was reduced from $\frac{6}{12}$ to $\frac{6}{60}$, and the field had become worse in the right eye. In the left eye, however, vision had improved from $\frac{6}{60}$ to $\frac{6}{24}$, and in the left lower temporal quadrant of this eye the field had improved. He asked when operation should be advised, if at all.

The PRESIDENT said the mere diagnosis of the presence of a pituitary tumor did not constitute a sufficient reason for operation. In illustration he quoted the case of an officer

who in 1916 has a large pituitary tumor and marked evidence of dyspituitarism. After much consultation it was decided not to operate, but to administer pituitrin. Some eight years later the sight had not got worse, but if he ceased the pituitrin he was worse until it was resumed. In another case in which no operation was done, there was some improvement in vision after a time, the likely explanation being that growth was proceeding in another direction, so that pressure on the chiasma was diminished.

Mr. M. S. MAYOU did not agree that what the President said applied in all cases. In one of his own, Professor Harvey Cushing, when in this country, did a de-compression operation, with much benefit. The patient had acromegaly, and that disease progressed, but the operation relieved the pressure on the chiasma.

Mr. FRANK THOMAS referred to a case of the same kind which he treated with small doses of pituitrin and thyroid, but without benefit. When seen, the sight of one eye had been quite lost, and in the other there was complete temporal hemianopia. The dose was then increased to 10 grs. thrice daily for a month, and the two lost fields were restored, the vision remaining at $\frac{6}{6}$. After a time a further increase in the dosage had to be made, until 15 gr. thrice daily was being taken. He was then able to resume his duties as postman for a year. After that he went downhill, and died.

Other cases were also shown.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. DAVID H. WEBSTER, Secretary.

MEETING OF OCT. 20, 1924. DR. BEN WITT KEY, CHAIRMAN.

Dr. MARTIN COHEN presented a case of **Bilateral Metastatic Carcinoma of the Choroid.** The left eye became involved twenty-two months after the removal of one breast for adeno-carcinoma. Two months later the right eye became involved. In each case a rapidly developing new growth appeared in the macular region, and in each eye the tension has been below normal.

DISCUSSION: Dr. WEEKS: Some years ago I saw a similar case in a woman sixty years of age, in which the growth in the eye was recognized before the general disease. I found a flat tumor and removed the eye. Diagnosis of metastatic carcinoma was made, and four months later the patient died of carcinoma of the lung. The condition in the eye was secondary.

Dr. COHEN: These cases differ from the usual sarcoma of the choroid by the ocular tension being minus and the condition being often bilateral.

Dr. MARTIN COHEN presented a case of **Lipemia Retinalis.** This patient had diabetes, the urine showed five per cent. sugar and a trace of acetone. He developed xanthoma diabeticum, and his blood cholesterol rose to about 1.200. The eye condition remained normal for about three years, after which time lipemia retinalis developed in both eyes, the condition being typical. Regulation of diet, and the use of iletin caused this lipemia to clear up in forty-eight hours. Dr. Cohen thinks that this condition is due to the fat content of

the blood rather than to the cholesterol. Dr. Cohen thanked Dr. Killian for conducting the chemical analyses in this case.

Dr. ALEXANDER DUANE read a paper on **Diplopia Observed in Various Types of Paralysis.** (Lantern Slide Demonstration.) He said that the existence of a paralysis was demonstrated by (a) a lagging of one eye (or overshoot of its fellow), (b) a characteristic screen deviation, and (c) a characteristic diplopia, each of these signs progressively increasing in some one of the six cardinal directions of the gaze. Of these signs the first and second were the most authentic, but the third was useful as corroborative evidence, especially in cases of slight paralysis where the other signs might be hard to demonstrate. He pointed out the special importance of the four corners of the field in diagnosticating paralysis of the vertically acting muscles. He also laid stress on the fact that the picture of diplopia in paralysis often differs from that given in the text books, in the following particulars: (1) The amount of diplopia does not correspond to the amount of deviation; (2) The primary diplopia (when the sound eye fixes) is not always smaller than secondary diplopia (when the paretic eye fixes); (3) The lateral deviation and (4) The torsion in paralysis of an elevator or depressor is by no means always according to the rule. These facts were illustrated by a series of lantern slides showing the diplopia as actually observed in paralyses of the individual muscles and in combined paralyses.

DISCUSSION: Dr. WOOTTON: In the traumatic paresis of the superior rectus, why not advance the superior rectus instead of tenotomizing the inferior oblique? It is a very simple procedure.

Dr. WHEELER: Could Dr. Duane give us some suggestions in tenotomy of the inferior oblique? Is there danger of getting too much effect, and does it make a difference where you cut the muscle? When should one excise a portion?

Dr. LAMBERT: In your case of advancement of the externus in which there followed a paresis of the internus, could this not be explained by the advanced muscle producing a check movement?

Dr. DUANE: In answering Dr. Wheeler's question I might say I have never attempted to graduate the amount of effect.

In tenotomy of the inferior oblique I usually remove part of the muscle as failures are due to a band left behind. I usually try to get all the effect I can. With regard to Dr. Lambert's question, the explanation is quite plausible in some cases. As in the case reported by the doctor, the check effect was evident, but in the case under discussion the paresis of the internal rectus did not occur until fifteen months after the operation. The check effect is one reason for not advancing the superior rectus because it produces a diplopia in the lower nasal field where it is most inconvenient.

Dr. TYSON: What is the average number of degrees of correction?

Dr. DUANE: It varies very considerably due to difference in muscular power, where there is a marked up-shoot in adduction it is most satisfactory.

Dr. M. URIBE TRONCOSCO demonstrated a new "Gonioscope" and read a paper on **Gonioscopy and Its Clinical Applications,** with lantern slide illustrations. It is a microscope and a periscope combined and gives magnifications of 10 and 20 diameters; it is used in conjunction with a Koeppe contact glass. A small electric lamp supplied by a dry battery contained in the handle, gives the necessary illumination.

The tube of the microscope is at right angles with the handle and is always held in front of the patient; the rays of light coming from the angle and parallel to the iris surface, are deviated by a square prism and sent to the observer's eye in front. The microscope is revolved on its axis for the examination of the angle all around the limbus. Focussing is made by moving the tip of the instrument back and forth from the eye.

The author described in a systematic way the structures of the normal angle. Beyond the border of the iris, the ciliary body is visible with the gonioscope, as a dark brown stripe, limited outside by the sclero-corneal trabeculum covering Schlemm's canal. This appears normally as a grayish band, and only in a few cases contains blood. Outside, the inner part of the sclera is seen as a white crescent, whose breadth changes in pathological conditions. The concave dome of the cornea and then the optical section of this membrane, which by an optical illusion looks like a microscopical

cross section, complete the picture. When a coloboma exists the ciliary processes and the orbiculus ciliaris as far as the ora serrata can be observed with the gonioscope.

Dr. TRONCOSCO also presented a review of the changes seen with this instrument in cases of iritis, glaucoma, and other conditions affecting the structures about the iris root.

DISCUSSION: Dr. SCHOENBERG had had the opportunity of using this instrument, and thinks that it needs further development, by being made stereoscopic and of greater magnification with a more intense light. A filtered light might bring forth some other points. This instrument opens up a new field.

Dr. TRONCOSCO does not consider a stationary instrument with great magnification to be practical. His aim was to have a handy instrument with sufficient magnification for the routine examination of the angle.

BOOK REVIEWS.

III.—**Errors of Accommodation and Refraction.** By Dr. Ernest Clarke, London. Fifth edition, 251 pages. Price, $3.50. William Wood and Company, publishers, New York, 1924.

The fifth edition of this well-known book has been thoroughly revised and many chapters re-written. In the twenty years since its first publication repeated revisions have served to eliminate all unnecessary or impractical material, and to have added the newer methods of proved value, so that now it is one of the most concise and useful books on this subject in the English language.

The chapters on elementary optics are especially good, and his refractive methods are clear and sound. In hyperopia he uses atropin in all cases up to 15 years of age, and when it does not too greatly inconvenience the patient, up to 30 years of age. He deducts I. D. from his atropin findings, which is somewhat more than most Ophthalmologists in this country deduct.

In Myopia he gives the full correction, and believes that the constant wearing of this correction will usually prevent further advance of this disease.

Because of his well-known views on Eye-Strain—that minor errors of refraction and minor degrees of muscle imbalance may lead either directly or indirectly to nearly all the diseases that man is heir to, including all the infections of the eye, cataract, pulmonary tuberculosis and insanity,—Dr. Clarke advocates the use of glasses in many cases where they would not ordinarily be given in this country, for here there is a growing tendency to leave off glasses whenever possible. Nevertheless, the book is a trustworthy guide to refraction and

deserves greater popularity in this country than it now enjoys. A. H. T.

IV.—**Diseases of the Eye.** By Dr. Geo. E. de Schweinitz, Professor of Ophthalmology, University of Pennsylvania, Philadelphia. Tenth edition, revised. 865 pages. Publishers, W. B. Saunders Co., Phila., 1924. Price, $7.

The tenth edition of this well-known textbook, follows the tradition established by preceding editions by careful revision and addition of those advances made in Ophthalmology during the last 3 years. These include new methods of examination, new diseases and new operations. The many features which make this our foremost American textbook are so well known that their repetition is unnecessary and in its new edition it will readily maintain its preëminent position.

A. K.

V.—**Die Tuberkulose des Auges** (Ocular Tuberculosis). By Dr. Arnold Lowenstein, Professor in the German University at Prague. 26 illustrations. 84 pages. Published by Urban & Schwarzenberg. Berlin and Vienna, 1924, Marks 2.40

The importance of tuberculosis as a cause of eye diseases has slowly made its way and it is to-day believed that no part of the eye escapes; in the author's opinion 50% of uveal diseases are tuberculous. The first 28 pages are devoted to an illustrated description of the clinical manifestations of ocular tuberculosis. The remainder is taken up with treatment. In this will be found a full description of light treatment, which depends principally on the intensity of the light and acts through active hyperæmia. The source of the light may be the sun, Finsen's light, the arc-lamp (Koeppe), Bach's quarz light and finally X-rays and radium. Specific treatment must be preceded by a correct diagnosis of the eye condition. This requires a careful general examination of the patient, clinical experience and the diagnostic tuberculin reaction. Tuberculin therapy is then fully described with a review of the literature, giving the opinion and results obtained by authorities in this field.

Dr Lowenstein's monograph is a valuable contribution to

the subject and will be found especially useful for those interested in tuberculin therapy. A. K.

VI.—**Manual of the Diseases of the Eye.** By DR. CHAS. H. MAY, New York. Eleventh edition, revised. 445 pages. Published by W. Wood & Co., New York, 1924. Price, $4.00.

Dr. May's excellent little book again appears in a new edition, revised and with additions to keep it up to date. The revision applies particularly to Uveitis, Chapter XI, and Disturbances of Motility, Chapter XXV, the latter with the assistance of Dr. J. H. Dunnington. The size of the book has not been increased and it will undoubtedly continue to fulfil admirably a want which its many editions has shown to be definite. A. K.

VII.—**Helmholtz's Physiological Optics.** An English translation edited by PROFESSOR JAMES P. C. SOUTHALL, Columbia University. Published in 3 volumes by the Optical Society of America, 1924. Price, $7.00 per volume. Orders should be sent to Professor F. K. Richtmyer, Secretary of the Optical Society, Ithaca, N. Y.

It is a great satisfaction to announce the translation into English of Hemholtz's classical work, which thereby becomes accessibly to a greater circle of readers and serves to stimulate and aid further study and investigation in this fundamental field. The translation was made possible by the Optical Society of America, and in particular through the efforts of one member, Mr. Adolph Lomb; it was resolved to celebrate the hundredth anniversary of the birth of Helmholtz by the publication of this translation. The committee in charge decided upon a translation of the 3rd German edition as it contained not only the original Helmholtz text but, recent additions in the physical, physiological and psychological portions by such eminent investigators as Gullstrand von Kries and Nagel. The American editor Professor Southall, Professor of Physics at Columbia, in the preface gives a list of about 12 collaborators who have helped in the translation. Some new and original matter has been added to this English translation which consists in (1) a chapter on Ophthalmoscopy by Gullstrand, (2) several special contributions by V. Kries and

(3) an article by Dr. Christine Ladd-Franklin on her color theory and related matters. A number of footnotes and a few references to the more recent literature are added.

Lenbach's celebrated portrait of Helmholtz and the prefaces of the three German editions are the beginning of Volume I, then after an **Anatomic Description of the Eye** follows the main part of this volume, namely, **The Dioptrics of the Eye** with the additions to each chapter in the form of Appendices by Gullstrand. One of these on Ophthalmoscopy is not found in the German 3rd edition but is taken from Gullstrand's "Einfuhrung in die Methoden der Dioptrik des Auges des Menschen," Leipzig, 1911, pp. 55–90. The translation is true, the style is clear, the typography and illustrations are excellent, in short a fitting presentation of this remarkable book, which reflects credit on both editor and publisher.

A. K.

NOTICES.

Convention of English-Speaking Ophthalmological Societies

To be held in London, July 14, 15, 16, 17, 1925

under the auspices of

The Ophthalmological Society of the United Kingdom and its Affiliated Societies

President, E. Treacher Collins
General Secretary, C. B. Goulden, O.B.E.

COMMITTEE FOR SCIENTIFIC BUSINESS AND PUBLICATION

Chairman, Sir John H. Parsons, F.R.S., C.B.E.
Secretary, R. Foster Moore, O.B.E.

COMMITTEE FOR GENERAL ARRANGEMENTS AND ENTERTAINMENTS

Chairman, Sir William Lister, K.C.M.G.
Secretary, J. F. Cunningham, O.B.E.

COMMITTEE FOR FINANCE AND REGISTRATION

Chairman, Sir Arnold Lawson, K.B.E.
Secretary, Leslie Paton.

The Ophthalmological Society of the United Kingdom, together with its affiliated Societies, has decided to hold in London from July 14th to 17th, 1925, a Convention of English-speaking Ophthalmological Societies and Associations, all members of which are invited to attend.

Letters have been sent to the Presidents of such bodies in the different parts of the British Empire and the United States of America inviting them to appoint official delegates.

On the evening of Monday, July 13th, before the opening of

the Convention, a Reception will be held at the Royal College of Surgeons of England.

Four mornings, two afternoons and one evening will be devoted to the Scientific work of the Convention.

The Bowman Lecture will be delivered by SIR JOHN PARSONS, C.B.E., F.R.S., on the afternoon of Thursday, July 16th.

On the Wednesday evening there will be a Symposium on the subject of "The Evolution of Binocular Vision," by the following selected speakers: SIR CHARLES SHERRINGTON, O.M., G.B.E., P.R.S., SIR ARTHUR KEITH, F.R.S., SIR FREDERICK MOTT, K.B.E., F.R.S., PROF. ELLIOT SMITH, F.R.S., and PROF. S. E. WHITNALL.

"Microscopy of the Living Eye" has been chosen as a subject for discussion. It will be opened by DR. GORDON BYERS, Montreal; DR. ARTHUR J. BEDELL, Albany, U.S.A.; MR. HARRISON BUTLER; and MR. BASIL GRAVES. Those wishing to take part in it, to read any paper or exhibit any specimen at the Convention should send in their names to the Secretary of the Scientific Business Committee, MR. R. FOSTER MOORE, 91, Harley Street, London, W.1, not later than four weeks before the meeting.

One afternoon of the Convention will be set apart for a visit to Institutions of Ophthalmic interest.

There will be a Museum of Drawings, Instruments, Pathological and Anatomical Specimens, to which all members of the Convention are invited to send exhibits.

At a General Meeting of the Convention the re-establishment of International Ophthalmological Congresses on the pre-war basis will be considered.

On Tuesday evening there will be a Reception at the Royal Institution by its President and Council.

A Garden Party will be given by SIR WILLIAM and LADY LISTER on Wednesday afternoon.

On Friday evening there will be a Reception at the Wellcome Historical Medical Museum by the President of the Convention.

The official Banquet of the Convention will be held on the Friday evening.

On the Saturday following the meetings of the Convention there will be Excursions to Oxford and Cambridge.

Local Committees connected with Affiliated Societies in various parts of Great Britain will assist those desirous of making more extended excursions.

The proceedings of the Convention will be conducted in accordance with the following rules and by-laws of the Society:

RULE 13.—"The Council shall decide upon all questions relating to the reception of communications, and shall have power to submit any paper to referees."

RULE 16.—"No paper shall be accepted if it has been published elsewhere. No paper shall be read before the Society unless a copy has been sent to the Secretaries at least four weeks before the meeting, together with an abstract suitable for immediate publication in the journals."

BY-LAW 2.—"No communication to the Society shall occupy more than twenty minutes, and in the subsequent discussion of it no member shall speak more than once, or for more than ten minutes without the special permission of the Chairman."

BY-LAW 3.—"All communications accepted by the Society become the property of the Society."

All communications must be typewritten.

The registration fee for those who are not members of the Ophthalmological Society of the United Kingdom will be £2, and for ladies accompanying members of the Convention who wish to become Associate Members, 10/-.

Every member of the Convention will receive a copy of its *Transactions* when published.

Thomas Cook & Sons have been appointed Official Transportation Agents, and their services will be placed fully at the disposal of Delegates for the issue of travel tickets, reservation of hotel accommodation of any grade desired, transfer and insurance of baggage, issue of travellers' cheques or letters of credit. A circular giving particulars of hotel charges in London during the period of the Convention, and other useful

information, will shortly be issued to registered members of the Convention.

It will facilitate the arrangements which are being made if those wishing to attend the Convention will as soon as possible send their name and address to MR. LESLIE PATON, 29 HARLEY ST., LONDON, W1, giving the name of the English-speaking Ophthalmological Society to which they belong and the name of ladies accompanying members who wish to become Associate Members.